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WHAT THE GENERAL PRACTITIONER SHOULD KNOW ABOUT OPHTHALMOSCOPIC EXAMINATIONS
Frederick A. Davis, M.D.

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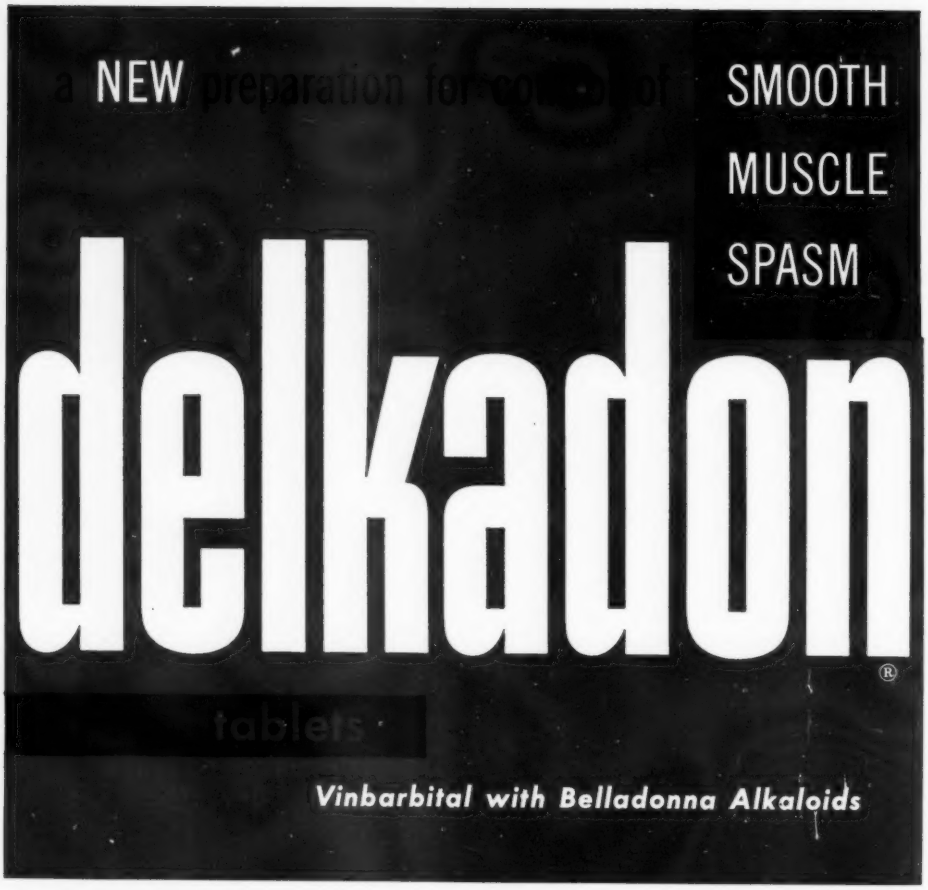

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What the General Practitioner Should Know About Ophthalmoscopic Examinations

FREDERICK A. DAVIS*

UNIVERSITY OF WISCONSIN MEDICAL SCHOOL, MADISON

I HAVE long been interested in the teaching of ophthalmoscopy to undergraduate medical students. From the beginning of our teaching of the clinical branches at the University of Wisconsin Medical School, instruction in the use of the ophthalmoscope has been part of the course in physical diagnosis, at first a limited part but gradually becoming more extensive in subsequent years. The aims and purposes of our course are to instruct the student in the mechanics of the use of the ophthalmoscope and to acquaint him with certain fundamentals in diagnosis which might encourage him to continue the use of the instrument in his routine clinical work. The scope of this course is limited in the main to a study of the normal fundus, with later emphasis upon the more practical aspects of ophthalmoscopy which are important for the general physician.

Unfortunately, the time allotted for instruction in ophthalmoscopy in most of our medical schools is so short that it is virtually impossible to give the student more than a brief introduction to the subject. It is my belief that little progress can be made toward a more general use of the ophthalmoscope by the general practitioner until the student receives more instruction in its use during his regular undergraduate medical course. It is easy to outline the many fundus changes which are important for the general physician, but unless he has acquired considerable knowledge concerning the normal fundus and its variations and makes frequent use of the ophthalmoscope in his routine work, it is unlikely that he will develop sufficient confidence in his interpretation of the things he sees to obtain much benefit from fundus examinations. When studying the fundus, that wise saying, "We see only that which we know," is especially applicable, for we can look long and see little if we do not know how to interpret the things we see.

In my opinion the general practitioner should use the ophthalmoscope in his daily work, if we can but teach him how to use it. He should regard it as a useful adjunct to a complete study of a case, just as he would a blood count, or the use of an otoscope for inspection of the tympanic membrane, though it must be admitted that the technic of ophthalmoscopy is more difficult to acquire. Far too many general practitioners utterly disregard the ophthalmoscope and thereby overlook valuable information which is readily available to them in the study of their cases. This was particularly true of those physicians of a generation or more ago.

*Department of Ophthalmology, University of Wisconsin Medical School.

Some internists and most neurologists have long recognized the importance of the ophthalmoscope in the routine study of their cases. Many have acquired skill in its use and proficiency in the interpretation of fundus changes equal or superior to that of many ophthalmologists.

With the perfection of the modern electric ophthalmoscope the long and tedious practice which formerly was necessary to visualize the fundus has been largely eliminated. Skill in the use of the instrument can be acquired by anyone if he is willing to make the effort. I would like to emphasize from the outset, however, that there is no easy, quick road to mastery of ophthalmoscopic diagnosis. A thorough understanding of the structure of the tissues which make up the fundus oculi, some knowledge of the underlying disease processes which manifest themselves in these parts, and painstaking daily use of the instrument are essential for those who would derive satisfaction from ophthalmoscopic examinations.

It is obvious that there should be limitations in the use of the ophthalmoscope for general practitioners, since many pathologic changes encountered in the eye fall exclusively within the sphere of the ophthalmologist. I refer to various diseases involving the media of the eye, such as cataract and uveitis, and many diseases of the fundus which are largely ophthalmic problems. Many other diseases, however, are but local manifestations in the eye of common general diseases which are frequently seen by the general practitioner. Before entering upon a description of the technic of ophthalmoscopy let us first consider some of the basic rules which should govern everyone who uses the ophthalmoscope.

CONDITION OF MEDIA AND SIZE OF PUPIL

First, to obtain a satisfactory view of the fundus, the media must be clear. By that I refer to the cornea, aqueous, crystalline lens, and the vitreous. If there should be clouding of these structures from various causes, difficulty will be encountered in visualizing the fundus and consultation may be necessary.

Second, the size of the pupil is of great importance, especially when one is not skilled in the use of the ophthalmoscope. Should a clear view of the disk and retina prove impossible through the undilated pupil, a weak mydriatic such as euphthalmine, 3 per cent, or ephedrine, 5 per cent, may be used to relax the sphincter. A complete and thorough study of all parts of the fundus is usually impossible unless the pupil is dilated. These drugs produce a moderate dilatation of the normal pupil in fifteen to thirty minutes, although at times two instillations may be required if the iris is rigid. Their action is not excessive or prolonged and it can be quickly neutralized by the use of a drop of a miotic, such as pilocarpine, 2 per cent, or eserine, 0.5 per cent.

One should bear in mind that careless or indiscriminate dilatation of the pupil in subjects of 40 years or over may, in rare instances, precipitate an acute rise in intraocular pressure, such as is seen in acute glaucoma, in an individual already the subject of the disease in its early or latent stage, which has been unrecognized. Therefore, it is always wise to use a counteracting miotic at the conclusion of the examination whenever the pupil has been dilated for diagnostic purposes.

TECHNIC OF EXAMINATION

A few practical points concerning the technic of ophthalmoscopic examinations may be of assistance to those inexperienced in the use of the instrument.

Two methods of examination are employed by ophthalmologists—namely, the indirect and the direct. The indirect method permits a much more extensive view of the fundus at one time, without changing the position of the examiner's head. However, the image is much smaller or less magnified, and it is inverted. I shall omit any description of indirect ophthalmoscopy, since, in my opinion, this procedure is too complex for the general practitioner. It is of value to ophthalmologists who become proficient in its use.

The direct method, utilizing the electric self-luminous ophthalmoscope, is the simplest and most easily mastered technic yet devised. The image is erect and greatly magnified, so that fine details are easily recognized. I think it helpful for both patient and examiner to be comfortably seated in a dark or semi-dark room. The examiner should sit on an easily movable stool on the patient's right side when examining the right eye, and on his left side when examining the left eye. The examination is easier if the examiner's and the patient's heads are on approximately the same level. The patient should be told to look straight ahead or slightly to the nasal side, directing the gaze of the eye not under examination at some object, such as a white disk of paper pasted upon the opposite wall of the room.

The ophthalmoscope should be held vertically in the examiner's right hand before his right eye when examining the patient's right eye, and in the left hand before the left eye when examining the patient's left eye. With the instrument held close to his own eye the examiner should approach the patient's eye, at the same time steadying the patient's head by placing his hand on the back of the head or neck. The light from the ophthalmoscope is directed into the patient's pupil a few inches from the eye until a fundus-glow is seen. The examiner then continues to approach the eye as closely as possible without touching it, whereby details of the eye ground will come into view. If the examiner has a moderately high refractive error, he should wear his glasses, but they mechanically interfere somewhat with the examination and may be discarded if desired.

Figure 1. Direct ophthalmoscopy (from Duke-Elder, Textbook of Ophthalmology; C. V. Mosby Co., 1940-41).

After the retina and retinal vessels come into view, one should follow the larger vessels to the point where they converge, viz., the optic disk or papilla. The disk is carefully scrutinized, then the surrounding retina, above and below, in and out, should be observed. It will be found helpful to adopt a systematic routine of study of the retinal vessels, starting with the superior temporal artery and vein, then the inferior temporal artery and vein, followed by the superior and inferior nasal vessels. In this way, one is less likely to overlook important changes in these vessels. If the patient has a high refractive error, a clear image may be possible only through adjusting the proper lens in the revolving disk of the ophthalmoscope to bring the retina into focus. This may be difficult or impossible when a high degree of astigmatism is present, for the image may then be blurred or indistinct. The May ophthalmoscope, Mayo model, is a very satisfactory instrument and is simple to operate.

OUTLINE OF BASIC FUNDUS CONDITIONS

I suggest that the general practitioner familiarize himself with the appearance of the fundus in the following five basic conditions:

1. He should know the normal fundus and its common variations.

2. He should be able to recognize abnormal blurring, congestion, swelling, or choking of the papilla, which is commonly seen in optic neuritis (papillitis) and in choked disk (papilledema).

3. He should be able to recognize pallor of the optic disk and varying degrees of atrophy, conveniently designated grades I, II, III, and IV.

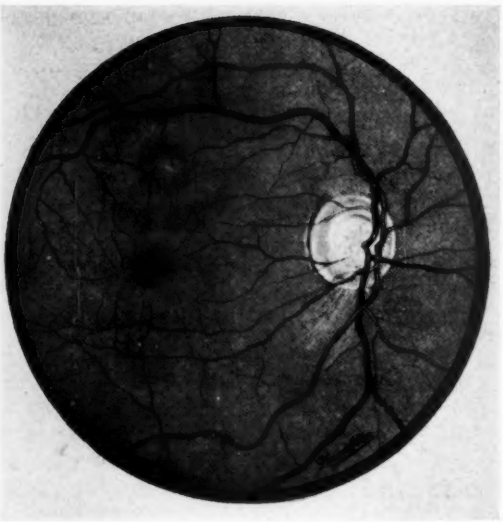

Figure 2. Normal fundus (from "The Anatomy of the Eye and Orbit" by Eugene Wolff, The Blakiston Co., Philadelphia).

4. He should be familiar with true glaucomatous cupping of the nerve head and be able to differentiate it from physiologic cupping.

5. He should be able to recognize abnormalities in the retinal vessels, particularly constriction or narrowing, spasm or obliteration, irregularities in caliber, tortuosity, beading, changes in color, arteriovenous compression, and hemorrhages and exudates in the retina.

Retinal hemorrhages and exudates frequently occur in the course of various general diseases, such as diabetes, hypertensive cardiovascular and renal diseases, the toxemia of pregnancy, etc. Their site, source, and character are varied, and the general practitioner may at first find it difficult to explore all their implications. It is important, however, for him to be able to recognize their presence; thereafter he may seek assistance concerning their nature and significance from someone more thoroughly trained in ophthalmoscopic interpretation.

THE NORMAL FUNDUS

There are many variations in the appearance of the normal fundus as seen with the ophthalmoscope. Some knowledge of these variations is essential for the interpretation of the ophthalmoscopic image as seen in both the normal and pathologic fundus.

The fundus picture is naturally dependent upon the anatomic structure of the various parts which make up the background of the eye. Therefore it may prove helpful to review briefly the anatomy of this region and some phases of the embryologic development.

EMBRYOLOGY AND ANATOMY

As you will recall in your study of embryology, primary optic vesicles appear early in embryonic life as outpouchings from the lateral aspect of the forebrain, the cavity of the vesicles being directly continuous with that of the forebrain. Subsequent invagination of the vesicle occurs, producing the secondary optic vesicle, or optic cup. The outer layer of cells of the cup, which are made up of neural ectoderm identical with that which lines the forebrain, becomes enormously thickened from subsequent multiplication and differentiation of the cells, to form the major portion of the adult retina, while the inner wall remains thin and finally resolves itself into a single layer of pigmented cells to form the pigment epithelium of the retina.*

The layer of pigment epithelium has a very important influence upon the ophthalmoscopic appearance of the fundus. Since the retinal layers which lie anterior to it are transparent, the amount and character of the pigment in the cells largely determines the many variations which are encountered in the normal and in the pathologic fundus. More extended reference to this will be found in the description of the normal fundus. *Pari passu* with these changes, the stalk or pedicle through which the optic cup is attached to the forebrain becomes

*In later stages, when the eye is fully developed, the designation "inner and outer" layers is reversed, to correspond to their relative position with relation to the center of the eye, e.g., the pigment epithelium becomes the outer layer, and the remaining layers become the inner layers.

Figure 3. Forebrain and optic vesicles of embryo rabbit (from Von Hippel, E., in Axenfeld, Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923). Left shows wide open primitive stalk. Right shows partial closure of the stalk.

Figure 5. Embryonic eye of the shrewmouse (preparation of Professor Kolmer of Vienna). Specimen shows a more advanced stage than Figure 4. The layer of pigment epithelium is well developed. Nerve fibers can be seen extending back through the primitive optic stalk: also remnants of the hyaloid artery. Thick inner layer has shrunk away from outer pigment in preparation. Original cavity of optic vessel remains as a potential space and, therefore, throughout life becomes more easily separated in retinal detachment.

constricted, with final obliteration of the cavity of the vesicle and gradual elongation of the stalk to form the framework of the optic nerve. With subsequent growth of the surrounding mesoderm the outer coats of the eye ball (sclera and choroid) are laid down, as well as the enveloping dura, arachnoid, and pial sheaths of the optic nerve and brain.* Nerve fibers originating in the ganglion cells of the retina extend back through the substance of the developing optic stalk to the midbrain, thereby forming this part of the optic nerve. Fibrous septa later extend into the nerve separating the nerve fibers into bundles.

Figure 4. Optic cup of the rabbit (from Axenfeld, after Von Hippel, Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923). Note the cup is continuous with the cavity of the forebrain, the primitive optic stalk being open.

It is thus seen that the retina is but an expanded portion of the brain itself, and the optic nerve a fiber tract similar to those of the central nervous system. The nerve fibers of the tract arise from the ganglion cells of the retina and extend back to the brain. The anatomical structure of the individual nerve fibers differs

*The more complex changes incident to the formation and closure of the fetal ocular cleft or fissure are here omitted for brevity and simplicity of presentation.

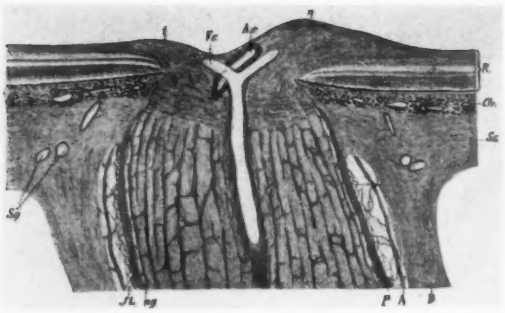

Figure 6. Longitudinal section through optic nerve, left eye (diagrammatic, from Axenfeld, after R. Graef, Textbook of Diseases of the Eye; Gustav Fisher, Jena, 1923). D: dura, A: arachnoid, P: pia, JL: intervaginal lymph-spaces, N: nerve fibers nasal to nerve fibers temporal side, Ac: central artery, Vc: central vein, Sg: scleral blood vessels.

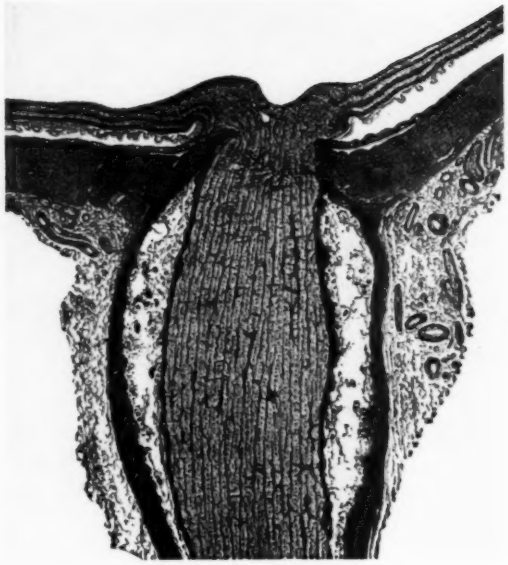

Figure 7. Choked disk from brain tumor (from Roemer, P., Textbook of Ophthalmology; translation of M. L. Foster; Rebman Company, 1917). Note swelling of the disk and distention of the intervaginal spaces of the sheath of the nerve.

from that of cerebrospinal nerves, being similar to that of the white matter of the brain. The sheaths of Schwann are absent, and the supportive framework is neurological.

The surrounding sheaths of the optic nerve—the dura, arachnoid, and pia—and their corresponding intervaginal spaces are continuous with those which envelop the brain. The dural sheath of the nerve passes over into the scleral coat of the eyeball, blending with its outer fibers. The thinner arachnoid likewise joins with and ends in the sclera. The pial coat hugs the nerve more closely and, in addition, sends prolongations into the body of the nerve, thereby contributing to the formation of the fibrous sepata which separate the nerve fiber bundles. It also enters into the formation of the lamina cribrosa or the perforated opening in the sclera and choroid, through which the nerve fibers pass in their passage to and from the retina. These sheaths, together with their accompanying subvaginal spaces, form what might be called an optic nerve canal which ends blindly at the level of the lamina cribrosa and sclera.

The subdural and subarachnoid spaces are capable of distention, especially the latter, both experimentally and clinically. Changes in intracranial pressure are readily transmitted down the subvaginal spaces to produce swelling or choking of the optic disk, so commonly seen in various types of brain lesions. The lamina cribrosa is recognized microscopically by numerous fibers of connective tissue which appear to cut in transversely across the axis of the nerve. These fibers, as stated above, consist of connective tissue from the sclera as well as the inward prolongations of the pia.

THE PAPILLA OR OPTIC DISK

The optic nerve is divided into a nonmedullated and a medullated section. The medullated fibers end approximately at the lamina cribrosa which bridges the opening in the foramen opticum sclera et choroid. The nonmedullated section of the nerve is that portion lying internal to the lamina cribrosa. It is sometimes further divided into a retinal, choroidal, and scleral portion; these areas correspond to the coats of the wall of the globe through which

Figure 8. Microscopic section of optic nerve and papilla (Weigert Stain) (from Guist, G.). Note dark staining of myelinated fibers of the nerve up to the lamina cribrosa, and decrease in size of the nerve internal to the lamina.

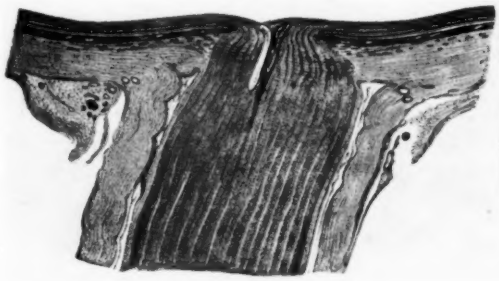

Figure 9. Microscopic section through normal optic nerve (diagrammatic) (from Adam, C., Ophthalmoscopic Diagnosis; translation of M. L. Foster, Rebman Co., 1913). The ophthalmoscopic appearance of this disk would appear uniformly pink or red with no cupping.

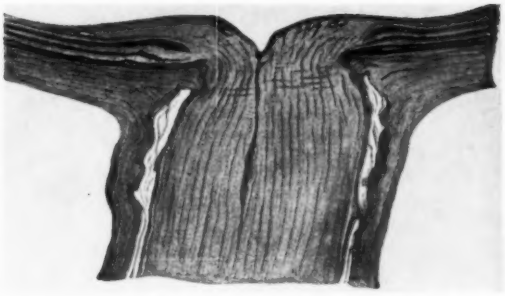

Figure 10. Small funnel-shaped excavation or cupping of the disk (diagrammatic) (from Adam, C., Ophthalmoscopic Diagnosis; translation of M. L. Foster, Rebman Co., 1913).

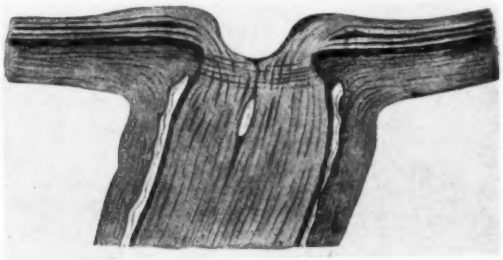

Figure 11. Deep physiologic cup (diagrammatic section) (from Adam, C., Ophthalmoscopic Diagnosis; translation of M. L. Foster, Rebman Co., 1913).

the optic nerve passes. I describe this region in some detail, since that part of the nerve which lies anterior to the lamina cribrosa makes up what we see with the ophthalmoscope and what we designate the disk, optic nerve head, or papilla. The lamina cribrosa is frequently visible at the bottom of the disk.

The foramen or opening in the sclera and choroid is approximately 1.5 mm. in diameter and varies slightly in form and contour from round to oval. The disk or intraocular portion of the nerve head which is visible with the ophthalmoscope is likewise round or oval and is made up of the nerve fibers which, beyond its borders, are spread out upon the inner surface of the retina. The surface of the disk varies. It may be smooth and flat, or it may show a funnel or a cup-shaped depression.

The flat papilla or disk is one in which the opening of the scleral canal is of such size that the nerve fibers completely fill up the foramen opticum sclerae and, therefore, little or no depression is present. This anatomical condition produces the reddish-pink disk seen with the ophthalmoscope, sometimes producing an appearance known as a pseudoneuritis.

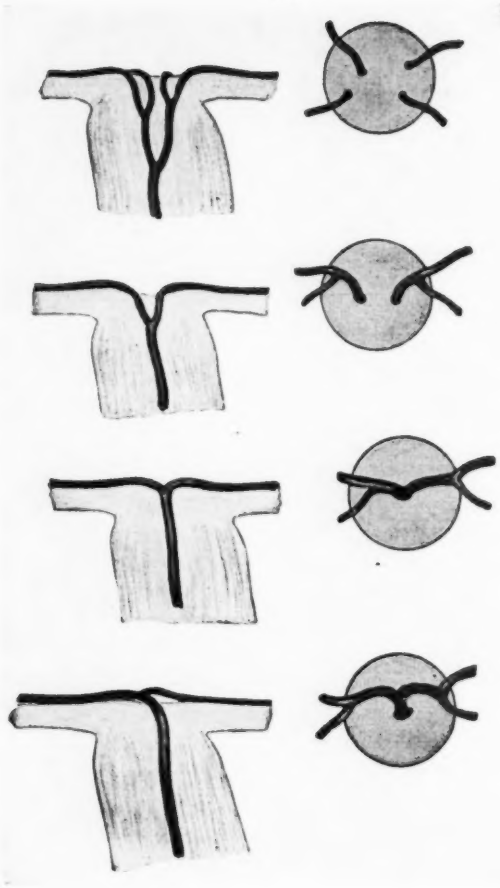

Figure 12. Schematic drawing of varied distribution of retinal vessels on the disk (from Adam, C., Ophthalmoscopic Diagnosis; translation of M. L. Foster, Rebman Co., 1913). When the vessel divides within the nerve the second and third divisions appear on the disk.

The excavated disk is one in which there is a funnel-shaped, cuplike depression in the center or, more frequently, slightly to the temporal side of the disk, commonly called the physiologic cup. This funnel- or cup-shaped depression occurs because the optic nerve fibers do not completely fill up the foramen opticum sclerae or hole in the scleral wall. They cling to the wall of the canal as they spread out upon the retina, and thus a central depression may remain. At times the cup is quite large and deep, though it does not reach the margin of the disk in the normal disk. The abrupt change in the size of the optic nerve as it enters the lamina cribrosa also contributes to the cupping, since the nerve becomes much smaller at this point owing to loss of the medullary sheathing of the individual nerve fibers; thus the bulk of the tissue which fills the scleral opening is reduced. The lamina cribrosa is thus visible at times at the bottom of the cup, as seen with the ophthalmoscope. Atrophy of the embryonic papilla of Bergmeister may also contribute to the formation of the physiologic cup.

The central retinal artery and vein reach the retina through the center of the nerve and are seen at the side of bottom of the cup if it is present; otherwise they appear to spring from the center of the nerve head or slightly to the nasal side. The main branches may divide within the nerve, or in the cup, or on the surface of the disk.

Knowledge of the anatomic variations in the tissues about the margins of the disk is essential for a thorough understanding of the ophthalmoscopic appearance, which may vary considerably within normal limits. In most eyes the layers of the choroid and retina surrounding the disk end at the disk margins. The marginal outlines are thus usually sharply defined, but in some instances the choroid and retina may stop a little short of the actual margin of the disk and thus a white circle, ring, or crescent may be seen just outside the pink area of the disk itself. This is called a scleral ring or crescent, and it appears because the overlying choroid and retina are absent in this area; thereby the border tissue becomes visible.

At times a black pigmentation is seen about the disk as a complete circle but more frequently as a crescent. This is due to the arrangement of the pigment epithelium of the retina. It may be unusually thick at this point, or it may project slightly beyond the overlying retinal layers and beyond the choroid. Since the retina and nerve fibers are transparent, the

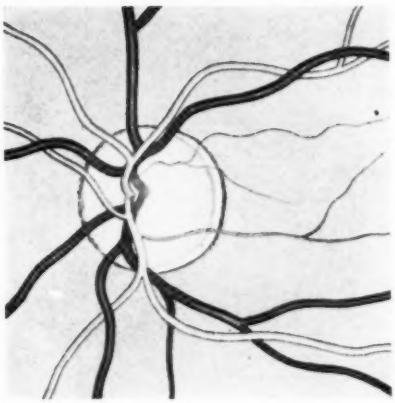

Figure 13. Scleral crescent with pigment ring (from Axenfeld, after Elschnig, A., Textbook of Diseases of the Eye: Gustav Fischer, Jena, 1923).

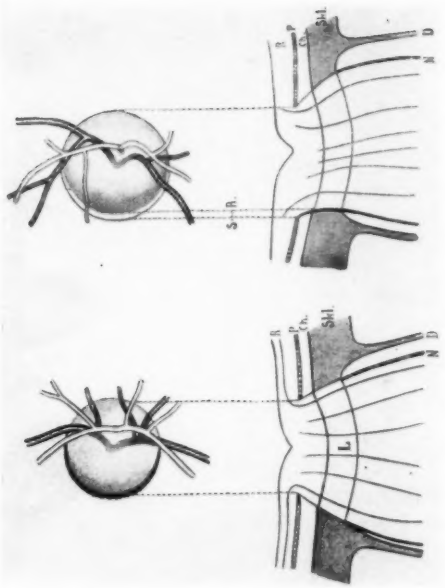

Figure 14. Diagrammatic drawing showing pigment crescent (bottom) and scleral crescent (top) (from Axenfeld, after Elschnig, A., Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923).

pigment shines through these structures and consequently it appears black. At times, more extensive white or pigmented crescentic areas of a pathologic nature, known as conus, border the disk. This type of crescent is frequently seen in high myopia.

Sometimes the nasal side of the disk is blurred, while the temporal side shows a wide white crescent. This is due to elongation of the globe and a stretching or displacement of the retina whereby it is pulled over and conceals the disk nasally, while on the temporal side the border tissue and the sclera become exposed and appear white. This condition is commonly referred to as traction and distraction or supertraction crescents.

THE RETINA AND CHOROID

It is not intended to enter into any detailed description of the histologic structure of the retina and choroid, since it is not essential for the general practitioner. No doubt most of you in your student days memorized the names of those 10 complex retinal layers for final examination in anatomy. The study of the structure of this delicate, transparent, filmlike coat, approximately 0.5 mm. in thickness, which lines the inner surface of the posterior part of the eyeball is a fascinating field for the ophthalmic histopathologist. However, anyone who undertakes ophthalmoscopic examinations will obtain greater satisfaction from them if he has some knowledge of the retinal architecture; without this knowledge he will have little appreciation of what he sees.

As previously described in the development of the eye, the retina may be divided into two main divisions: (1) the layer of pigment epithelium, and (2) the remaining nine layers which lie anterior to it (Figures 5 and 18). Since the inner layers are transparent in the normal state, they are virtually invisible when viewed with the ophthalmoscope, except for the retinal vessels which traverse them. Therefore, it is the layer of pigment epithelium, made up of a single row of hexagonal pigmented

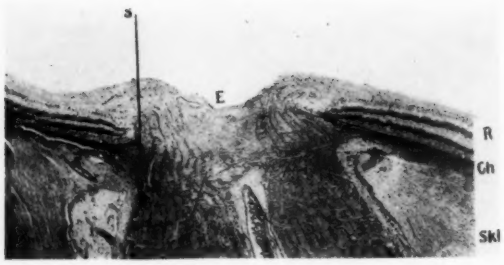

Figure 15. Microscopic section of normal nerve head (from Guist, G.). Showing histologic basis for pigmented crescent or ring bordering disk. R: retina, Ch: choroid, Skl: sclera, E: physiologic excavation, S: layer of pigment epithelium projecting inward beyond other retinal layers.

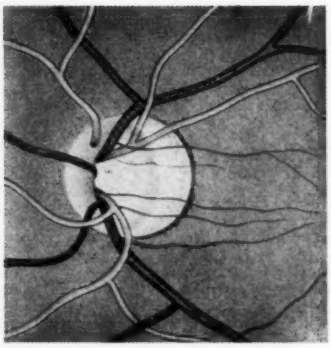

Figure 16. Temporal conus in myopia (from Axenfeld, after Elschnig, A., Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923). Traction and distraction crescents due to stretching incident to the myopia.

cells, which largely determines the variations encountered in the normal fundus. If the melanin pigment which is present in these cells is heavy and densely packed, nothing can be seen of the adjacent choroid which lies beneath it. However, if the pigment is lightly distributed or is absent, as in the case of albinism, the choroid, and at times the sclera, may become visible.

The choroid is a thin, soft, brown coat which measures approximately 0.25 mm. in its thickest area. It is extremely vascular, being largely made up of vessels and pigmented cells. The chief function of the choroid is to provide the right blood supply to the outer layers of the retina, viz., the rods and cones. The blood vessels of the choroid and the intervening masses of dense pigment are frequently visible with the ophthalmoscope when the pigment epithelium of the retina is thin or absent.

OPHTHALMOSCOPIC APPEARANCE OF THE NORMAL FUNDUS

The appearance of the normal fundus may vary considerably in different individuals, depending upon age, complexion, race, etc. It is, therefore, important that you become familiar with these variations in order that you may not confuse them with pathological changes.

Systematic writers frequently describe the normal fundus under three different types, to which I should like to add a fourth: (1) the uniform stippled fundus, (2) the tasselated fundus, (3) the albinotic fundus, and (4) the mixed-type fundus.

These variations depend, in a large degree, upon the amount, type, and distribution of the pigment in the choroid and the pigment epithelium of the retina. The intensity of the pigmentation usually follows the color of the hair and skin of the individual, varying with the blond and brunette types.

1. The uniform stippled fundus has a uniform red color, varying somewhat with the amount of pigment present. The color may be a deep, brownish-red, or slate gray in the dark-skinned races, e.g., the negro, or it may be a yellowish-red in the yellow races. The pigmentation is usually more dense surrounding the disk and in the macular region.

In younger subjects, especially hyperopes, fleeting shotlike reflexes from the surface of the retina may flit across the surface of the retina as the light is moved about over it. The light is so reflected because of inequalities in the surface of the retina owing to the underlying vessels. The stippled type of fundus is produced by the dense and uniform pigmenta-

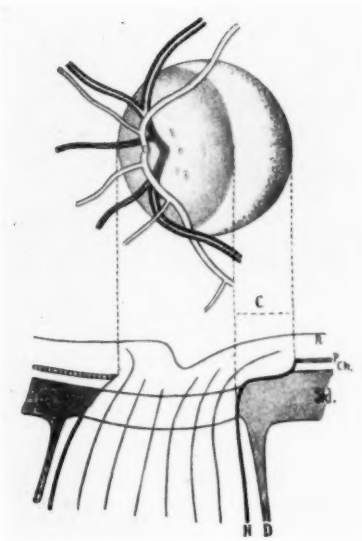

Figure 17. Temporal conus (from Axenfeld, after Elschnig, A., Lerbuch und Atlas der Augenheilkunde; Gustav Fischer, Jena, 1923). C: conus, R: retina, P: pigment epithelium, Ch: choroid, Skl: sclera.

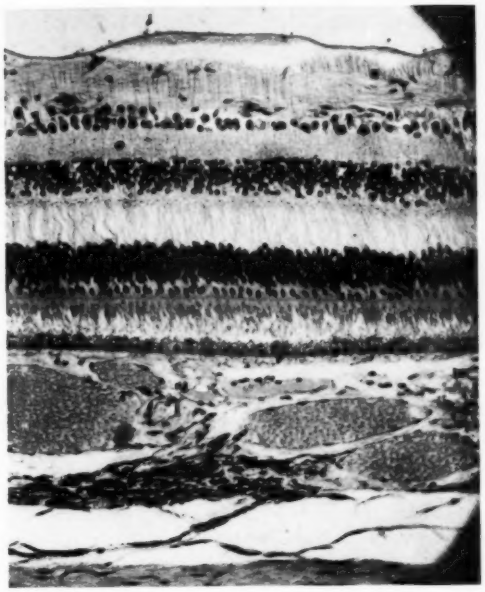

Figure 18. Section through the normal human retina and choroid. Preparation of Dr. E. E. MacEwen (from a photograph supplied by Dr. James Shields). Layers of retina: 1. Pigment epithelium; 2. Rods and cones; 3. External limiting membrane; 4. Outer nuclear layer; 5. Outer molecular layer; 6. Inner nuclear layer; 7. Inner molecular layer; 8. Ganglion cells; 9. Nerve fiber layer; 10. Internal limiting membrane.

tion of the pigment epithelium, which conceals the underlying choroid.

2. The tasselated or tigroid fundus is frequently encountered and appears as a dark brown or gray mottling on a bright red-streaked background. It occurs when the pigment epithelium of the retina is but lightly pigmented. The masses of choroidal pigment appear as dark, irregular islands on a bright red background. The bright red areas are made up of choroidal vessels which interlace, anastamosing freely and running in every direction. Extreme types of the tigroid fundus might easily be mistaken for choroiditis by those who are unfamiliar with it.

3. The albinotic fundus is characterized by the complete absence of pigmentation in both the retina and the choroid, so the varied pattern of the choroidal vessels becomes visible against the white or yellowish background of the sclera. The choroidal vessels are distinguished from the retinal vessels by their uniform, bright pink color, by the absence of a light reflex in the vessels, and by their abundant anastomoses. The vessels lie deeper than the retinal vessels, and they have a flat, bandlike contour. At times albinism may be incomplete or partial, with only certain areas of the retina involved. Although the albinotic fundus is usually classified as a normal type, high myopia often accompanies it, and frequently the visual acuity is markedly impaired.

4. The mixed-type fundus is very frequently encountered. In this type the uniform stippled condition prevails over the larger part of the central area and surrounds the disk, but toward

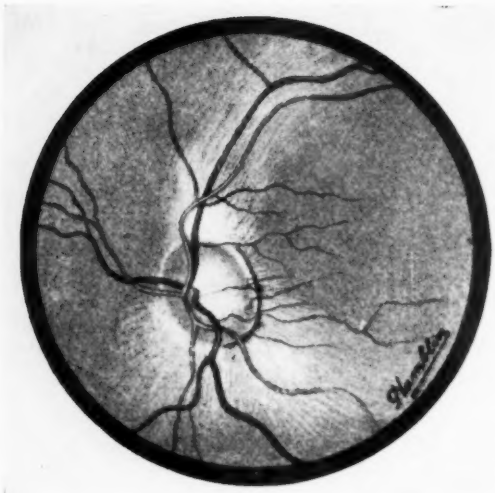

Figure 19. Opaque (medullated) nerve fibers (from Mann, Ida, Developmental Abnormalities of the Eye; Cambridge University Press, England.) Used by permission of The Macmillan Company, New York.

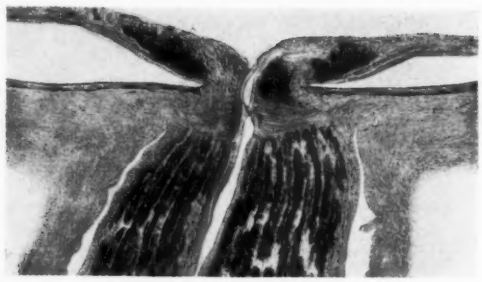

Figure 20. Medullated nerve fibers (Weigert Pal stain) (from Axenfeld, after Elschnig, A., Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923). Note medullated fibers stained dark.

the periphery of the fundus the choroidal vessels are frequently visible owing to a thinning of the pigment in this area. This condition is present in quite a high percentage of the fundi of normal individuals seen in routine fundiscopic examinations following refraction.

THE macula is located in the line of the visual axis at the posterior pole of the eye. Its center, the fovea, lies about 3.5 mm. or 2½ disk diameters to the temporal side of the disk and slightly below the horizontal meridian. It is the most sensitive part of the retina and its structure is so arranged as to permit maximum visual acuity only in this area. It is devoid of retinal blood vessels, being nourished by the underlying choriocapillaris of the choroid. The macula is often shown in textbooks as an oval area somewhat larger than the disk itself. Anatomically this is correct, but ophthalmoscopically the entire macular area is rarely visible. Occasionally in young subjects a bright oval reflex, like a halo, is seen at the periphery of the macular area, but full details of this region are visible only with special illumination, such as red-free light. With this method, the yellowish coloration from which it derives its name, macula lutea, can be seen.

The usual ophthalmoscopic appearance of the macula consists of a small white spot surrounded by an area more deeply pigmented than the rest of the retina. This white spot is nothing but a reflex from the curved surface of the macula at the fovea. Its form and shape change, therefore, as the light from the ophthalmoscope is moved in different positions. The sharp contour and brightness of the reflex usually diminishes with advancing years. The macular area can be best observed if the patient is directed to look at the light in the ophthalmoscope, providing the pupil is well dilated. If the pupil is not dilated, this procedure is not satisfactory, since the pupil will contract sharply, and the light reflex from the cornea may prove annoying or may make it impossible to view the macula under these conditions. The inexperienced may have to search for the macula and may find it helpful to note that the fine terminal retinal blood vessels which surround this area point toward it but end before reaching it.

THE DISK OR PAPILLA

The optic disk or papilla is the point of greatest interest in the fundus. It may be located by

following the larger vessels to the point where they converge or emerge on the surface of the retina. It can best be observed if the patient is directed to look slightly in, or nasalward, since it lies to the nasal side of the visual axis and is, therefore, out of line when the examiner looks into an eye whose gaze is directly forward.

WHEN examining the disk it is well to keep in mind the following points: (1) form and size, (2) color, (3) margins, (4) surface level (excavation or protrusion), and (5) blood vessels.

1. The disk is usually round or slightly oval. There is, at times, an apparent variation in size, the disk being smaller in high degrees of hyperopia (farsightedness) and larger in high degrees of myopia (nearsightedness). Its shape may be distorted in high degrees of astigmatism when it is frequently elongated and somewhat blurred or indistinct, occasionally so much so as to be classified as a pseudoneuritis.

2. The color of the disk is variable. It is usually light pink in color and somewhat paler than the surrounding retina. This varies, however, depending upon its contrast with the color of the surrounding retina. It has a paler pink hue in the dark stippled fundus of the brunette, and a reddish-pink color in the pale fundus of the blond. The temporal half of the disk is somewhat paler than the nasal, in part due to the presence of a physiologic cup. Since the nerve fibers are spread apart in the cup, a part of the lamina cribrosa becomes visible at the bottom of the cup. The nerve fibers in the temporal half of the disk are finer and fewer in number, since they are limited to a group of fibers which make up the papillomacular bundle which supplies the macula. The nasal half of the disk has a deeper pink color owing to the presence of a greater number of nerve fibers in this area; consequently, the greater number of fine blood vessels or capillaries, which accompany the nerve fibers, impart a deeper pink hue to it.

3. The disk margins are usually fairly sharply defined, but the superior, inferior, and nasal borders are less distinct than the temporal due to the greater number of nerve fibers which pass over these areas. The arrangement of the nerve fibers of the temporal half of the retina is such that most of them by-pass the macular area and thus reach the disk above and below by a peculiar arcuate bending which crowds the fibers at the superior and inferior borders of the disk. At times the blurring produced by this is so marked as to raise the question of a neuritis, which must be ruled out by further study of the visual acuity and fields. When the physiologic cup is entirely absent the disk may appear congested, with a blurring of all the disk margins, thus producing yet another condition known as pseudoneuritis.

Medullated nerve fibers rarely extend beyond the lamina cribrosa and are seen ophthalmoscopically as intensely white patches adjoining the disk. They have a characteristic, silky, white, striated appearance, the outer edges resembling the fine hairs of a paint brush. They lie in the nerve fiber layer of the retina and frequently cover the adjacent retinal vessels. They remain stationary throughout life and produce no loss of visual function aside from an enlargement of the blind spot in the area involved.

4. The surface of the disk is usually level with that of the surrounding retina excepting only the area of the physiologic cup, which is depressed. The cup may appear as a slightly cone-shaped depression or it may take the shape of a sharp, deep, punched-out cup. When the cup is large and deep, the bundles of nerve fibers become visible at the bottom of the cup as gray spots in contrast to the intense whiteness of the lamina itself. This is due to the fact that the nerve fibers lose their myelin sheaths as they enter the lamina; they are more translucent and, therefore, reflect less light than the surrounding connective tissue.

If the physiologic cup is quite large, it may be confused with the pathologic cupping seen in glaucoma. The criterion for differentiation of the physiologic cup from that of glaucoma is that the former does not extend to the border

of the disk. Usually some normal, healthy nerve fibers intervene between it and the margin of the disk, which is recognized ophthalmoscopically by the light pink coloration of this tissue. The cup of glaucoma extends to the border of the disk at some point. At times, however, the physiologic cup has a shelving edge as it approaches the temporal margin of the disk and so resembles the cupping of glaucoma that other tests, such as plotting of the fields and blind spots and measuring of the intraocular pressure, must be used in order to establish its true nature.

The surface of the normal disk on rare occasions may be slightly elevated when the physiologic cup is absent. This condition arises when the opening in the scleral canal is so small that the fibers of the optic nerve completely fill up the opening or overcrowd it, thereby producing a piling-up or an elevation of this region. This type of nerve head is usually red in color and frequently may be mistaken for a neuritis, but careful study and proper understanding of these structural variations should easily establish its true nature. This condition is known as pseudoneuritis.

5. The retinal blood vessels enter the eye near the center of the disk, usually at the nasal side of the optic cup, and spread out over the retina, dividing into innumerable branches. Their walls are transparent, and therefore the blood column is visible as it courses through them. Arteries are recognized by their bright red color whereas the veins have a deeper wine-red hue. The arteries are narrower than the veins, being about three-fourths of the veins' width, and both show a slight streak which is largely due to a reflex from the blood column. The light streak from the arteries is broader and brighter than that of the vein.

The main branches of the retinal vessels lie in the anterior layers of the retina just beneath the internal limiting membrane. Finer branches extend into the inner portion of the deeper layers. No vessels are seen in the macula, since this area of the retina is nourished by the underlying choriocapillaris of the choroid.

The arrangement of the vessels on the disk is variable, depending upon the point at which the main stem divides. At times the vessel divides deep in the nerve stem and therefore appears on the disk as several subsidiary branches. The vessels usually divide on the disk into two main branches, superior and inferior, and further subdivide into nasal and temporal branches.

The vessels pursue a wavy course across the retina, with varying degrees of tortuosity. Tortuosity of the larger branches of both the arteries and the veins varies considerably in the normal eye, so it is not to be relied on as evidence of disease, as a rule, though there are exceptions to this.

Pulsation of the vein as it bends to enter the physiologic cup is generally visible with the ophthalmoscope. Pulsation of the artery does not occur in health but is seen in pathologic states, such as aortic regurgitation and glaucoma. The retinal vessels do not anastomose, so occlusion or thrombosis is followed by disastrous consequences, since there is no collateral circulation. The choroidal vessels, when visible, are readily recognized by their lighter uniform pink color and the absence of the light streak. They anastomose freely and follow no pattern but run in every direction. Their deeper position is easily recognized, since the retinal vessels lie anterior to them.

OPTIC NEURITIS AND CHOKED DISK

THE conditions in which optic neuritis may occur as an incidental complication are too numerous to discuss in the limited scope of this paper.

Optic neuritis may arise from inflammatory conditions within the eye or orbit itself, or in adjacent structures, such as the nasal sinuses. It is relatively common in certain affections of the central nervous system, especially disseminated sclerosis. It is occasionally observed in most of the acute infectious diseases. Syphilis, foci of chronic sepsis (teeth, tonsils, etc.), tobacco, alcohol, and a variety of metallic poisonings may at times attack the nerve in its intraocular or retrobulbar portion.

When the inflammatory process affects the nerve head or the papilla, it is commonly called papillitis; when it is confined to the trunk of the nerve, it is termed retrobulbar neuritis. Since retrobulbar neuritis rarely shows any ophthalmoscopic evidence of the acute inflammatory phase of the disease, I shall omit any extended discussion of it. A residual pallor of the disk, especially of the temporal half, which appears some time after the acute process has subsided, may be the only ophthalmoscopic change seen. Multiple sclerosis is responsible for the great majority of these cases and usually occurs in persons under thirty. It is typically unilateral and is sudden in onset; it causes a rapid loss of vision, and presents a normal-appearing nerve. I have recently seen a case, however, in a young woman, thirty years of age, who had a typical mild papillitis.

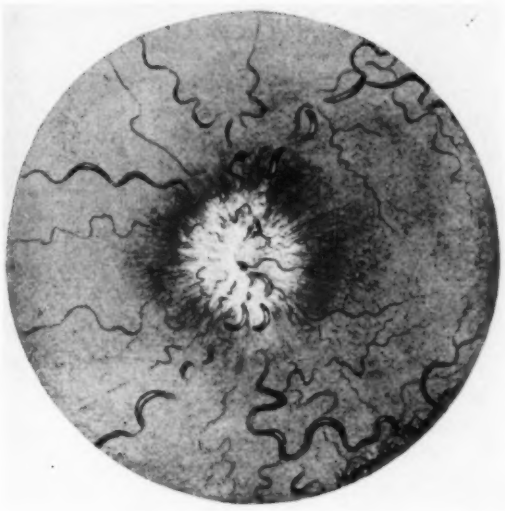

Figure 21. Papilledema (choked disk) (from de Schweinitz, G. E., Diseases of the Eye; W. B. Saunders Co., 1916). From a patient with tumor of the brain.

Intraocular neuritis or papillitis is a relatively common disease. The first ophthalmoscopic evidence of the disease is hyperemia of the disk. The margins become blurred and indistinct, and the veins slightly engorged. The hyperemia is rapidly followed by a mild degree of swelling, usually not over two to three diopters, though occasionally more. Flame-shaped hemorrhages appear on the disk and immediately around it, and at times edema and some exudates appear in the adjacent retina. The disease is usually preceded by marked reduction in central visual acuity. This may develop before there is any ophthalmoscopic evidence of the disease. The onset may be sudden or gradual. As a rule the disease is transient, and recovery may take place in a few weeks under proper treatment. In mild cases remarkable and complete restoration of vision may follow, with only slight residual pallor of the nerve. In others there is severe damage to the nerve fibers with marked optic atrophy and permanent loss of vision.

CHOKED DISK OR PAPILLEDEMA

The term choked disk or papilledema is used when swelling or protrusion of the nerve head arises from increased intracranial pressure. It is primarily a passive edema of the disk, unassociated with any inflammatory change; for a considerable period there may be no disturbance of visual function. Papilledema, therefore, has a different etiology from optic neuritis or papillitis, and a different pathology and symptomatology, although the appearance of the two states may be indistinguishable ophthalmoscopically in certain stages of the two affections. This is especially so either in the early stages of choked disk or in mild cases. Careful evaluation of various other associated symptoms may be necessary to arrive at a correct diagnosis. Early reduction in visual acuity, which at times may be profound, is characteristic of papillitis, whereas in papilledema the visual acuity may be but slightly affected for a considerable period of time.

Intracranial pressure arising from brain tumor is responsible for papilledema in a great majority of cases. Not infrequently it is a relatively late symptom, however. It may also develop in connection with various other le-

Figure 22. Papilledema in cerebral tumor (from Duke-Elder, after Paton, Textbook of Ophthalmology; C. V. Mosby Co., 1940-41).

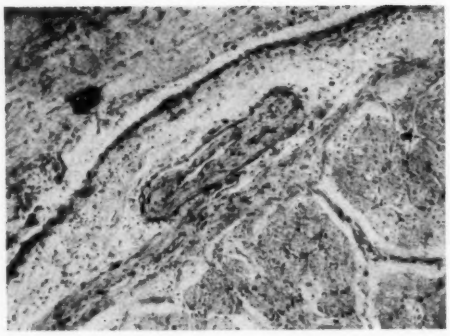

Figure 23. Papilledema (from Duke-Elder, after Paton and Holmes, Textbook of Ophthalmology, Vol. III; C. V. Mosby Co.). Showing compression of central retinal veins in the subarachnoid space.

sions, such as brain injuries, abscess, and meningitis. Toxic states, such as septic foci, may rarely be the underlying cause. I have observed a swelling of the optic disk of 7 diopters in a young person of 15, which receded rapidly after the removal of two abscessed teeth. This might be better classified as an optic neuritis, but the ophthalmoscopic appearance and symptomatology in some cases may be indistinguishable from that of choked disk. General diseases, such as the malignant phase of essential hypertension, produce marked swelling of the disk, though the exact mechanism of its development is still not completely understood.

As a rule the pathologic changes encountered in papilledema consist of simple edema in the region of the nerve head with swelling and infiltration of all the tissues with fluid. The vessels, particularly the veins and capillaries, are enormously distended, and hemorrhages are found on the disk and surrounding retina.

THE exact manner by which choking arises is still in doubt, though many theories have been advanced. Mechanical compression or strangulation of the central retinal vein as it leaves the nerve stem 10 to 15 mm. behind the globe, as described by Paton and Holmes, probably accounts for a considerable part of it. Accumulation of cerebrospinal fluid in the subvaginal spaces produces marked distention of this space, with subsequent compression of the nerve and the vein as it traverses the canal. The wall of the vein, being thin, may become compressed or collapsed, thereby retarding the return flow of blood from the retinal veins, with consequent edema and hemorrhage. Since the arterial wall is thicker, and the intravascular systolic pressure higher, it is more resistant to compression.

The clinical picture is quite characteristic when the process is in an advanced stage. The nerve head is enormously swollen and definitely protrudes forward much like a mushroom. It may be elevated as much as 9 or 10 diopters above the surrounding retina. There is increased redness, the vessels being swollen, particularly the veins, which are greatly engorged. The arteries are small and in places, may be completely concealed by the swollen and edematous nerve fibers.

The edema may finally extend to the surrounding retina, particularly toward the macular area, where white streaks may develop in the form of a fan-shaped figure. This arises from edema and degenerative products beneath the internal limiting membrane. It may resemble the star-shaped figure of malignant hypertensive retinopathy but it is less sharply defined

and does not, as a rule, completely surround the macula. Fluffy white spots may appear on the swollen disk owing to degeneration of nerve fibers and accumulation of serum and fibrin. The swelling of the disk may persist for a very long time, usually until the intracranial pressure is relieved by operation or until other pathologic states responsible for it have been removed. Subsidence of the swelling is usually slow except after cerebellar operations.

The degree of loss of visual acuity is quite variable. Frequently in the early stages and for a long period after the swelling is well established there may be little or no loss in visual acuity, with enlargement of the blind spot being the only change found. The reason for this is that the process is not a true inflammatory change but a simple passive edema. The nerve fibers, being quite resistant, may tolerate pressure for a considerable period of time without damage. However, in long-standing cases with increasing pressure, central vision gradually deteriorates, and later, with subsequent atrophy, considerable damage and contraction of the visual fields occurs.

I believe that it is important for the general practitioner to be familiar with the ophthalmoscopic appearance of papillitis and papilledema. Certainly in every patient suffering from persistent headache, nausea, and vomiting, a fundus examination should be made by the general physician who is usually the first one to see the case, since this classical triad is pathognomonic of intracranial pressure associated with brain tumor.

OPTIC ATROPHY

Atrophy of the optic nerve should be of especial interest to the general practitioner, since it may be the first sign of tabes. It may, however, be the result of a very wide variety of causes. Duke-Elder, in his recent text, has admirably classified the various types. I have briefly summarized his classification in Table 1.

TABLE 1

ETIOLOGICAL CLASSIFICATION OF OPTIC ATROPHY
(from Duke-Elder)

I. Consecutive atrophy secondary to retinal disease and destruction of the ganglion cells.
   A. As after chorioretinitis and other retinal diseases
   B. Degenerative, as is high myopia
II. Circulatory atrophy. Occlusion of central retinal artery; arteriosclerosis; pressure from calcified arteries
III. Pressure and traction atrophy
   A. Glaucoma
   B. Postpapilledema
   C. Arterial changes; sclerosis
   D. Bony pressure, in deformities such as tower skull
   E. Tumors of the optic nerve, in the orbit or cranium; Von Recklinghausen's disease. (Glioma of optic nerve or chiasm)
IV. Post-inflammatory
   A. Optic neuritis
   B. Metastatic infection; tubercle and syphilis
   C. Diseases of the central nervous system, as disseminated sclerosis, tabes, dementia paralytica
V. Toxic atrophy
   A. Endogenous toxins, e.g., diabetes, anemia
   B. Exogenous poisoning, e.g., tobacco, arsenic, lead, etc.
VI. Traumatic atrophy, e.g., avulsion and fractures in region of the optic canal
VII. Lebers, hereditary optic atrophy

It is my purpose to discuss but few of these, since it would lead us far afield, but a brief glance at Table 1 will serve to illustrate the great variety of diseases which may affect the optic nerve.

Optic atrophy is usually designated as primary or secondary, the primary type indicating that there has been no antecedent disease, whereas the secondary variety follows inflammatory episodes, such as optic neuritis, or choked disk, or other intraocular disease.

Simple or primary atrophy is the classical type which is associated with the late manifestation of neurosyphilis, especially tabes and taboparesis. In this the disks are grayish-white with sharply defined margins. In its final stages a saucer-like depression of the disk may develop due to shrinkage of the nerve substance. It appears many years after the primary syphilitic infection.

Failure of vision and pallor of the optic nerve may be the first signs of tabes. They may precede the onset of other signs of the disease by as much as twenty years, so that it occasionally falls to the ophthalmologist's lot to make the first diagnosis. However, the atrophy may appear later, after ataxia has developed. It always involves both nerves, though one may be af-

fected before the other. The atrophy develops slowly, with a gradual loss of vision. Vision may be surprisingly good, however, at times when the nerve appears quite white, a circumstance not infrequently found in other forms of optic atrophy. According to the present most popular view the atrophy arises as a result of a peripheral and interstitial neuritis produced by the treponoma of syphilis and its toxins which cause secondary degeneration of nerve fibers.

Although the classical description of the ophthalmoscopic appearance of the disk is gray or gray-green, it not infrequently is white, and the appearance is indistinguishable from that of any other primary atrophy. The retinal vessels are little affected in the earlier stages; later they become narrowed. The remainder of the retina may appear essentially normal for a time. Eventually thinning may occur from atrophy of the ganglion cells.

The general physician should never fail to study the fundi of patients with small, rigid pupils which are inactive to light but contract in accommodation—a characteristic of the Argyll-Robertson pupil—since optic atrophy is so frequently encountered with this symptom. Many other fundus changes due to syphilis might be mentioned but are really beyond the scope of this paper.

The physician should be on the alert for temporal pallor of the disk, commonly seen after repeated attacks of multiple sclerosis. He should also be familiar with the slight temporal pallor associated with the large physiologic cup, previously referred to, otherwise he may confuse this with pathologic changes. Ophthalmologists frequently describe the pallor of optic atrophy as grades I, II, and III, a somewhat rough but convenient method of indicating the degree of atrophy present.

CHRONIC SIMPLE GLAUCOMA

It may appear strange to some that I include the fundus picture of glaucoma among the "must" rules laid down as essential for the general practitioner. I insist that this is highly proper, for unless the ophthalmologist can enlist the aid of the general practitioner in the early recognition of this disease, there seems little hope of reducing the incidence of blindness resulting from it. A program of publicity for the lay public might help, but here again the practitioner would be of great assistance, since many apprehensive individuals will seek advice from him once they are aware of the danger of the disease.

In my experience blindness from chronic simple glaucoma seems to be by no means lessening. Approximately 12 per cent of all blindness is attributed to glaucoma, and in the upper age groups (over 45 years) the percentage is probably much higher, though accurate statistics are difficult to obtain.

How can the general practitioner help in this program for preservation of vision? If he makes daily use of the ophthalmoscope, he may learn to recognize the early stages of cupping of the optic disk which accompanies the disease. Although it is admitted the disease may be relatively advanced when cupping has become evident with the ophthalmoscope, a considerable measure of vision can nevertheless be preserved even at this late date if atrophy is not advanced. It is unquestionably too much to expect the general practitioner to become familiar with other signs which appear in the early stages of the disease, such as increase in intraocular pressure, shallowness of the anterior chamber, and the various changes in the visual fields, but I feel he can, with some training and effort, learn to recognize the glaucoma cup.

As a rule, patients with acute glaucoma consult an oculist, since pain in and about the eye is so acute and agonizing that they seek relief. However, the pain is at times referred to the temples, face, teeth, and other parts of the head, and is frequently accompanied by vomiting. I have known cases which were under treatment by the general practitioner for stomach trouble for a considerable time before it was discovered that the eye was involved. He should be on the alert for this fulminating type. However, the

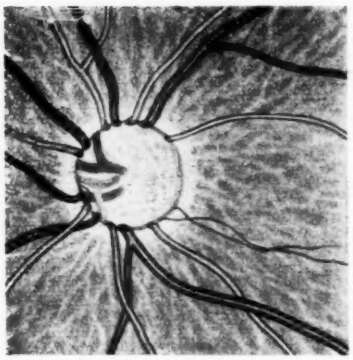

Figure 24. Glaucoma cup—total (from Axenfeld, after Elschnig, A., Textbook of Diseases of the Eye; Gustav Fischer, Jena, 1923). Note fundus is of the "tasselated type."

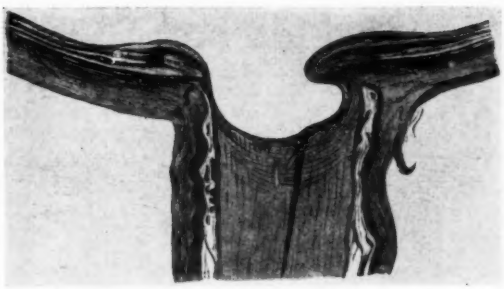

Figure 25. Deep glaucomatous excavation (from Adam, C., Ophthalmoscopic Diagnosis; Rebman Company, 1913). Note complete destruction of nerve head from intraocular pressure.

fundus usually cannot be visualized in acute glaucoma due to steaminess of the cornea, so the ophthalmoscopic examination may be unsatisfactory or impossible.

Chronic simple glaucoma, or glaucoma simplex, sometimes referred to as "compensated glaucoma," is frequently without symptoms of *any kind* for a considerable period in the course of the disease. This results chiefly from the fact that the rise in intraocular pressure is gradual thereby allowing the tissues of the globe to become adjusted to the pressure, and inflammatory symptoms do not develop. It is not at all uncommon for a patient to be brought to the office almost totally blind, except for a small telescopic field, unaware that he had the disease. To be sure he has been conscious of gradually failing vision (often mistaken for cataract), but frequently he has not been fully cognizant of this, since central acuity or the ability to read ordinary print may be preserved for a very long time, even though the disease is gradually progressing. This is due to the fact that the disease first affects the peripheral field, especially on the nasal side, with some other well-known peripapillary field defects, while the central vision is spared almost to the end. Optometrists who examine large numbers of patients for glasses usually are totally ignorant of fundus pathology except for some of the more recent graduates who are showing some progress in this regard. The general physician should, therefore, make every effort possible to increase his knowledge of the disease.

THE GLAUCOMA CUP

The cupping of the disk in glaucoma is, as a rule, easily recognized although there are borderline cases in the early stages in which it is impossible to differentiate it from the physiologic cup, and many other tests are then required to establish the diagnosis. The typical cup is relatively large. At first it occupies the central and temporal portions of the disk, but eventually it expands to involve the entire disk. The margins of the cup are usually sharply defined, and the sides are steep, so that it presents a definite punched-out appearance. Blood vessels which enter the cup are often pushed to the nasal side. They hug the sides of the cup and dip down beneath its overhanging edges to disappear partly from view as they curve around it. They reappear in the bottom of the cup and can be brought sharply into focus by adjusting the lenses in the ophthalmoscope. When the disease is not far advanced and the cup is relatively shallow, it presents a uniform gray color; as the cup deepens, however, atrophy

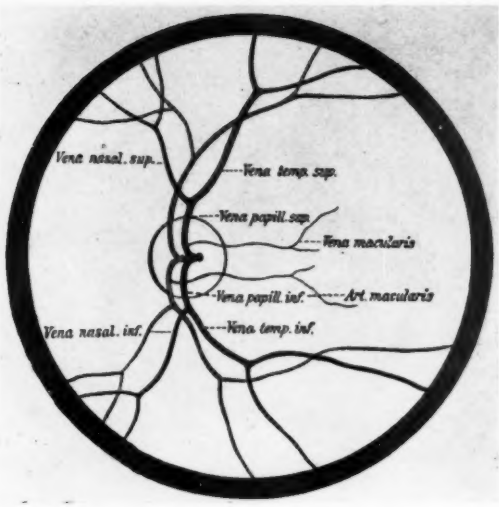

Figure 26. Schematic drawing of ophthalmoscopic appearance of retinal vessels (from Adam, C., Ophthalmoscopic Diagnosis; Rebman Company, 1913).

of the nerve fibers progresses, and the entire area becomes white.

When the increased intraocular pressure is of long standing, the pigment of the retina and choroid becomes thinner. This may be so marked immediately surrounding the disk that the sclera may be partly exposed. This condition is recognized ophthalmoscopically as a white circle bordering the disk, known as the glaucoma halo. This term should not be confused with the well-known colored halo observed by the patient when he looks at artificial lights at night. The latter occurs more frequently in the prodromal stage of acute glaucoma, but it also occurs at times in chronic simple glaucoma when the intraocular pressure rises sufficiently to induce edema of the cornea, which accounts for the phenomenon.

The retinal vessels may show only slight change. The arteries become somewhat narrower; the veins are frequently slightly engorged. Pulsation may appear in the central artery on the disk, though this is of little diagnostic significance since it occurs in a number of other conditions and, rarely, in healthy eyes.

RETINAL VASCULAR DISTURBANCES

The remainder of this paper will be devoted to various common retinal changes that are of interest to the general practitioner. Some of these changes will be discussed at length, since they are of great importance not only in ophthalmoscopy but in the diagnosis of other diseases.

Retinal vascular changes furnish important clues in the early diagnosis and prognosis of many diseases of interest to the general practitioner, particularly the cardiovascular-renal diseases. The diagnosis of such affections as arteriosclerosis and essential hypertension may be greatly facilitated by careful and painstaking study of the retinal vessels.

Some physicians consider information gained from ophthalmoscopic studies in these diseases to be of greater value than any other single diagnostic procedure. Certainly no other means of examination affords the direct visual evidence of vascular changes that is offered by the ophthalmoscope.

When viewing the retinal vascular system it is well to remember the extremely small size of the vessels. The caliber of the retinal arteries is such that they should be classified as arterioles. The diameter of the central retinal artery is about 0.25 mm., or 1/100 of an inch, but due to the magnification of the dioptric system of the eye the apparent size of the vessels when viewed with the ophthalmoscope is increased to approximately 1 to 1.5 mm., varying according to the refractive error of the eye. The veins being slightly larger and much darker in color are more readily visualized. The light streak which traverses the vessels is a striking feature, especially in the larger branches. It is brighter and wider in the artery than in the vein. The veins follow the course of the arteries in a rough way, though the vessels may cross and recross each other in their course.

In healthy individuals the contour of the vessels is usually perfectly smooth and regular, showing no variation in caliber, except that

Figure 27*a*. Normal vessels. Caliber of artery: average. Caliber of vein: average. Reflex stripe on artery: moderate. Lumen of artery: regular. Compression of vein at arterial crossing: none.

Figure 27*b*. Senile fibrosis. Caliber of artery: constricted. Caliber of vein: normal or constricted. Reflex stripe on artery: normal or mildly exaggerated. Lumen of artery: regular. Compression of vein at arterial crossing: none or mild. Sclerosis or choroidal arteries often associated.

Figure 27*c*. Signs of hypertension. Caliber of artery: uniformly constricted. Caliber of vein: normal or relatively dilated. Reflex stripe on artery: uniformly exaggerated. Lumen of artery: regular. Compression of vein at arterial crossing: moderate.

they gradually become smaller as they extend farther from the disk to the periphery of the retina. Moderate tortuosity of both arteries and veins is commonly seen in healthy individuals. At points where the vessels cross each other there is no deviation in their course nor is there any change in their size and contour. In diseased conditions, such as arteriosclerosis and hypertension, the vein frequently becomes indented when crossed by the artery, so that it shows a definite constriction and, at times, an apparent break in the regular course of the vessel.

RETINAL SIGNS OF AGING

With advancing years some fibrosis develops in the vessel walls. This is recognized by narrowing of their lumina, lessened translucency, and dulling of the light streak, especially of the arteries. This is not accompanied by any change in the regularity of the lumen of the vessels. Clinically, it is manifested by a general narrowing or attenuation of all the retinal vessels with little or no abnormal elevation of blood pressure. The choroidal vessels usually participate in this change to a certain degree, frequently recognized through the thinning retina of senility. If advanced arteriosclerotic changes develop, sclerosis of the choroidal vessels is strikingly visible through the ophthalmoscope as a white interlacing pattern bordering the pale pink outline of the vessels.

DRUSEN

Another rather common change in the retina of people of advanced years is known as *drusen*. These appear as small round or oval dots of a yellowish-gray (sometimes a light pink) color scattered over the fundus. At times they become quite prominent and may be lighter in color, surrounded by a more densely pigmented halo.

Anatomically drusen consist of small discrete elevations on the lamina vitrea of the choroid, just beneath the pigment epithelium. They are made up of a colloid material produced by some abnormal or excessive secretory action of the pigment epithelium, which is it-

self displaced by the substance as it accumulates. These drusen bodies or colloid deposits may at times be few in number, or they may be numerous. They are considered a normal accompaniment of senescence, and as a rule produce no defect in vision unless they are massed in the macular area, in which case a considerable reduction in central vision may follow. Occasionally they are seen in young people.

SENILE MACULAR DEGENERATION

Senile degeneration of the macula is relatively common in the aged and is associated with arteriosclerosis. The ophthalmoscope may show anything from a few scattered pigment dots to widespread degeneration in the macular and perimacular area. The remainder of the retina may appear essentially normal, aside from thinning and some signs of sclerosis of the choroidal and retinal vessels.

The macular degeneration arises from sclerosis of the choroidal vessels, particularly the choriocapillaris immediately beneath the macula. A reduction in central visual acuity, which may be profound, may be the only symptom for a considerable period, since involvement of the periphery of the choroid and retina may be slight or absent for some time.

ARTERIOSCLEROSIS AND HYPERTENSION

Ophthalmoscopic examination can be an important aid in the diagnosis of generalized arteriosclerosis and hypertension. It should be remembered, however, that the retinal vessels are but a localized area of the blood vascular system and they may not always depict the status of the vessels throughout the body. Duke-Elder stresses the well-known fact that vascular sclerosis of various types is rarely uniform, at one time being predominantly ocular, at other times cardiac, cerebral, or renal. However, statistics from many observers reveal a high incidence of retinal arteriosclerosis in generalized arteriosclerosis, varying from 40 per cent to 80 per cent. A similar situation is revealed in hypertension. Wagner reports 56.6 per cent retinal arteriosclerosis in 200 cases of hypertension, though generalized arteriosclerosis was not evident in 30 per cent of the patients showing retinal arteriosclerosis. Of the 200 cases 46.5 per cent showed general disease, but 20.4 per cent of these showed no retinal involvement, and 13.5 per cent had cerebral arteriosclerosis though no retinal changes were associated in 25.9 per cent of these.

Thus it will be seen that arteriosclerosis is a "capricious disease," as so aptly phrased by Duke-Elder in his masterly discussion of various phases of this complicated subject, "varying much in the extent of its manifestations all over the body, but most authors are agreed that *arteriolarsclerosis* is distributed regularly with great uniformity throughout all the organs. Moreover, while retinal sclerosis can give no safe indication of the *degree* of sclerosis elsewhere, its occurrence in the eye almost certainly indicates its presence generally."

Arteriosclerosis of the retinal vessels is manifested not alone by extreme narrowing of the vessels but also by changes in color of the vessels. The light streak of the artery may become extremely bright due to the increased reflection of light from the thickened medial coat of the vessel wall, which may finally advance to a state of hyaline degeneration at which point the color of the reflex takes on a coppery hue; they are then commonly described as "copper wire arteries." As the sclerosing process increases, the thickened walls of the arteries become more visible, and the vessels assume a silvery-white hue and are designated "silver wire arteries." Periarterial fibrosis may be added to this, occasionally producing "pipestem arteries," so called because of the encircling white coating. This is regarded by some investigators as evidence of previous retinitis in these areas. Irregularities in the lumen of the arteries may develop as a result of thickening of the vessel walls.

The finer terminal arterial twigs may become extremely tortuous, resembling a corkscrew. This may be particularly noticeable in the perimacular areas, where the small terminal branches point toward the macula, a condition well described by de Schweinitz. When the arteriosclerotic process is advanced, areas of

localized ischemia may develop in the retina, with resultant degenerative areas recognized ophthalmoscopically as small white "hard" spots, usually in the deeper layers of the retina in the central area. Small hemorrhages may also develop, appearing striate or flame-shaped when they lie in the superficial layers of the retina, or round and sharply defined when confined to the deeper layers.

These changes may be bilateral or unilateral and may remain unchanged for long periods of time, according to Foster Moore, who has furnished the most complete and thorough description of the condition. These changes have been designated "arteriosclerotic retinitis" by him. The disease runs a relatively benign course. Since an associated hypertension of varying degree may develop in the course of the disease, some authors attribute many of the changes observed to this and question the validity of Foster Moore's contention that the condition is primarily an arteriosclerotic process. However, it is seen quite frequently and does not have the characteristics of essential hypertension either from an ophthalmoscopic or from a general clinical standpoint. I have seen retinal changes of this type in the aged, whose blood pressure had never been elevated beyond that considered normal for their age group.

OCCLUSION OF THE CENTRAL ARTERY AND VENOUS THROMBOSIS

Occlusion of the central retinal artery by embolism or thrombosis may develop in the course of retinal arteriosclerosis, though other diseases may be a contributing factor. It is recognized by generalized pallor of the retina due to ischemia, marked constriction or disappearance of the retinal arteries, and the well-known cherry-red spot at the macula. The macula appears red due to the presence of the underlying choriocapillaris by which it is nourished, and it is, therefore, not affected by the retinal edema incident to the occlusion of the retinal artery.

Figure 28. Thrombosis of a branch of the central retinal vein (from Duke-Elder, after Parsons, Textbook of Ophthalmology; C. V. Mosby Co., 1940-41).

Thrombosis of the central retinal vein or of one of its branches may also develop in arteriosclerosis, especially when some added toxic factor is superimposed. Thrombosis of one of the branches of the main vein frequently occurs at a point of arteriovenous crossing when the vein has previously suffered some damage from constriction due to sclerosis of the adjacent artery or fibrosis in the connective tissue septa common to the vein and artery. These conditions, while frequently disastrous from a visual standpoint, usually are not of immediately grave prognostic significance so far as life is concerned, for many patients live for years after the vascular accident.

The ophthalmoscopic picture is quite characteristic and easily recognized. When the main central vein is involved, massive hemorrhages are seen scattered throughout the retina. The veins are much engorged and tortuous; the arteries are unaffected, though at times ob-

scured by accompanying retinal edema. White patches later appear in the retina, and at times a star-shaped figure may develop in the macula. In branch-thrombosis a similar picture is seen, limited to the area involved. Frequently the site of the rupture of the vein can be seen, usually at the point of an arteriovenous crossing.

DIABETIC RETINOPATHY

A retinitis (retinopathy) similar to that described as "arteriosclerotic retinitis" occasionally develops in diabetics. The ophthalmoscopic appearance is quite characteristic, consisting of small round or oval white degenerative spots, or so-called exudates, which lie in the deeper layers of the retina. Numerous hemorrhages, typically round or punctate, are scattered throughout the retina, chiefly in the central area. They may precede the development of exudates for a considerable period of time. They are sharply delimited as a rule, owing to their position in the deeper layers. Sometimes the white dots assume a circinate arrangement surrounding the macula when somewhat larger globular white areas become confluent.

There is still some controversy as to the pathogenesis of the retinal changes associated with diabetes, with some authorities contending that they are the result of arteriosclerotic changes which so commonly occur in diabetes. However, since typical diabetic retinopathy appears at times in young subjects who show no evidence of arteriosclerosis, the retinal changes are regarded by many observers as a distinct clinical entity which arises from metabolic disturbance incident to the diabetes. At times an associated hypertension may serve to complicate the typical retinal changes which are so characteristic of the disease.

HYPERTENSION

The fundus changes which are associated with various types of hypertension have assumed considerable importance in recent years due to the advance in our knowledge of this disease. The retinal changes which occur in arteriosclerosis and renal disease have been long known, and their grave prognostic importance in certain forms of kidney disease have been generally recognized.

Only in comparatively recent years has the hypertension per se, whatever may be its cause, assumed the major role which it is now accorded in the evaluation of cardiovascular-renal diseases. At the present time many authorities attribute the varied retinal changes, which at times accompany these diseases, solely to the associated hypertension.

ELWYN, in an article concerning the various fundus changes which may be observed in hypertension, has admirably summarized the basic conditions which may at times be responsible for the rise in blood pressure. I submit this summary in a somewhat abbreviated form.

FORMS OF HYPERTENSION
(from Elwyn)

I. Essential hypertension—benign phase
II. Malignant renal sclerosis, or the malignant phase of essential hypertension
III. Hypertension of renal origin
  A. Diffuse glomerulonephritis (acute, subacute, subchronic, and chronic)
  B. Rare forms of renal disease
  1. Amyloid kidney
  2. Unilateral renal hypoplasia
  3. Polycystic kidney
  4. Periarteritis nodosa with renal vascular changes
  5. Hydronephrosis
  6. Pyelonephritis
  7. Prostatic obstruction
IV. Hypertension of pre-eclampsia and eclampsia of pregnancy
V. Hypertension accompanying tumors of adrenal glands and basophilic adenoma of the pituitary gland (Cushing's syndrome)

It is evident from this classification that hypertension may occur in a variety of conditions. The subject is too extensive to attempt a description here of the varied retinal changes which may develop in the course of all of these diseases. Volhard, Fishberg, Wagener, and many others emphasize the importance of the hypertension per se, as the basic cause of the retinal changes which are encountered whether it is of essential, renal, or toxic origin. It may be seen, therefore, that at times a similar re-

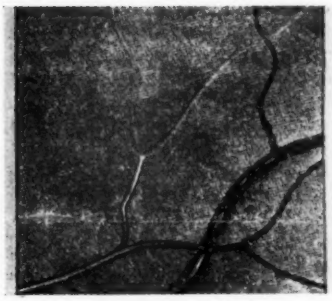

Figure 29a. Spasm of artery (from Gipner, John F., The Clinical Significance of Retinal Arterial Changes and Retinitis in Cardiovascular Renal Disease: New York State J. Med., August 1930). Artery: normal or sclerosed. Vein: normal. Caliber of artery: constricted at point of spasm. Lumen of artery: obliterated at point of spasm. Peripheral branches of artery: reduced to threads or invisible.

tinal picture may evolve in a variety of general conditions in which hypertension is the dominant characteristic. Differentiation or separation of some of these is therefore difficult and, at times, impossible.

Since the kidney is frequently involved, early or late, in most of these conditions, the retinal changes were formerly attributed to the associated albuminuria. Now it is well known that albuminuria has nothing to do with the retinal changes, so the old familiar term "albuminuric retinitis" has been discarded for the term "hypertensive neuroretinopathy." The latter is more descriptive since it indicates that the disease process arises from hypertension and that the retinal changes are degenerative rather than inflammatory in character. Because of limited space I shall confine my discussion of this subject to the retinal changes associated with so-called essential hypertension since they are by far more frequently encountered and, therefore, are of greater interest to the clinician and ophthalmologist alike.

ESSENTIAL HYPERTENSION

In defining the term "essential hypertension," Fishberg states, "the concept of essential hypertension includes those cases of chronic hypertension which neither clinically nor anatomically can be demonstrated to have evolved from antecedent inflammatory disease or urinary obstruction." This author, however, admits the definition is defective and further states "the very term 'essential hypertension' is a confession of ignorance and this is its chief virtue."

Duke-Elder gives a somewhat wider scope to his definition, viz., "Essential hypertension is a persistent and usually progressive raising of the blood pressure which represents the response of the vascular system generally to some underlying disorder, hereditary, metabolic, toxemic, infective, endocrine, or psychological, which probably causes primarily an overactivity of the vasomotor center by nervous or chemical pressor agencies."

A SATISFACTORY classification of the retinal changes observed in essential hypertension is difficult to offer since neither ophthalmologists nor clinicians agree concerning either the pathogenesis or the terminology of the disease. Likewise, description of the vascular changes encountered in retinal vessels vary with practically every writer on the subject. It is my purpose merely to point out certain commonly recognized signs in the retina and retinal vessels which may aid the general practitioner in establishing a diagnosis and following the progress of the disease.

The terms "benign" and "malignant hypertension" have been fairly generally used by some authors, particularly Keith and Wagener, though they are by no means universally adopted by all writers. The term "benign hypertension" refers to cases of persistent elevation of blood pressure which may extend over a period of many years and which may be, in the earlier phases, accompanied by few if any general symptoms and by virtually no visible changes in the fundus oculi.

Therefore, for a considerable period in the course of benign essential hypertension the retinal vessels appear normal, and ophthalmoscopic examination would be of assistance only for its negative value (Elwyn). Unless blood

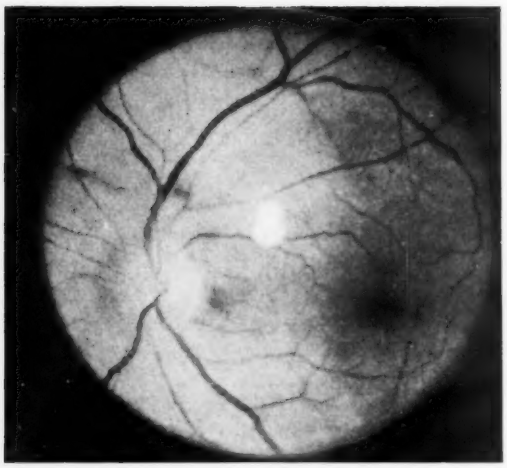

Figure 29*b*. Diffuse vasospasm of retinal arteries in hypertension (Grade II) (Author's case). Blood pressure 216/126. Urine negative.

Figure 31. Retinitis of malignant hypertension (third stage) (from Lillie, Cardiovascular Renal Disease; D. Appleton-Century Co.). Note extensive edema of disk; marked attenuation of arteries; numerous hemorrhages and exudates.

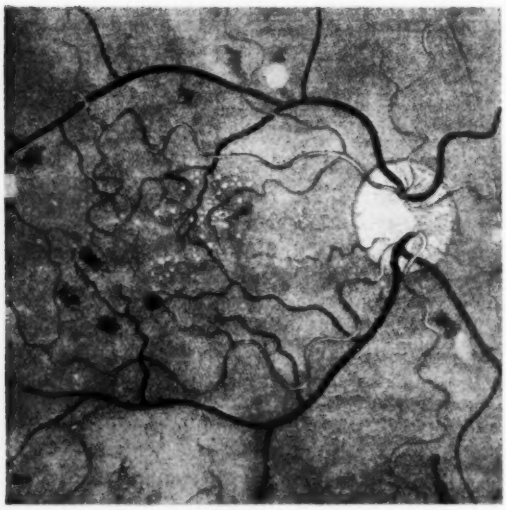

Figure 30. Retinitis of hypertension (severe benign type of Wagener) (from Gipner, John F., The Clinical Significance of Retinal Arterial Changes and Retinitis in Cardiovascular Renal Disease; New York State J. Med., August 1930). Arteriosclerosis of hypertension type; diffuse narrowing of arteries; exaggerated reflex; irregularities in caliber of terminal vessels; arteriovenous compression; cotton-wool patches; scattered hemorrhages.

pressure readings are made by the ophthalmologist as well as by the clinician, many of these cases will be overlooked.

In the later stages of benign hypertension important signs appear in the retina. These evidently are a result of wear and tear on the retinal vessels caused by the persistent hypertension which thereby induces varying degrees of vascular sclerosis. One of the most common signs encountered is narrowing of the arteries and indentation or deviation of the veins at arteriovenous crossings. Arteriovenous compression, or variations of it, may arise from many different pathologic changes in and adjacent to the artery. Fishberg states that it may be present at times without arteriosclerosis, especially in hypertension of recent or acute origin, such as is seen in toxemia of pregnancy and acute glomerulonephritis, when venous compression may be attributed to the increased rigidity of the overlying artery owing to turgidity incident to the increased blood pressure. This is sometimes called "Salus' sign," since he made some very thorough studies of it.

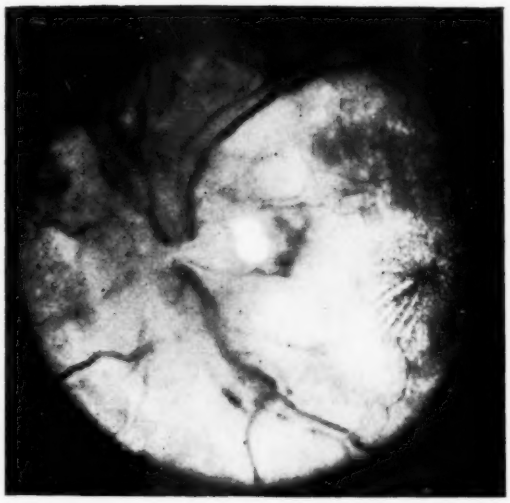

Figure 32*a*. Malignant neuroretinopathy (author's case). Photographs of the fundus of a patient with malignant hypertension (female aged 40; kidney function excellent; blood pressure 240/136). Note edema of the disk and retina; marked spasm of the retinal arteries; tortuosity of the terminal vessels; numerous hemorrhages and exudates; beginning star-shaped figure in the macula.

Figure 32*b*. Malignant neuroretinopathy (author's case). Photographs of the fundus of a patient with malignant hypertension (female aged 40; kidney function excellent; blood pressure 240/136). Note edema of the disk and retina; marked spasm of the retinal arteries; tortuosity of the terminal vessels; numerous hemorrhages and exudates; star-shaped figure in the macula, more advanced stage.

Most authors consider arteriovenous compression merely as evidence of sclerosis in the vessel walls which has been brought about by the persistent hypertension; it is, therefore, included under the heading "arteriosclerosis" in some classifications of the disease. In my experience, arteriovenous compression and variations of it are constantly seen in early hypertension. I seldom have failed to find a considerable elevation of blood pressure when its presence is marked. As a rule, the diastolic pressure is usually disproportionately elevated, recordings of 90 to 100 being quite common. Generalized narrowing of the retinal arteries is usually also present, and later, when the process is more advanced, irregularity in the caliber of certain vessels may appear. This condition apparently may remain relatively stationary for a considerable period of time, or it may gradually progress.

Usually if we are dealing with a true malignant type of hypertension, the retinal vessels show a generalized vasospastic contraction (see Fig. 27-c), variously described as angiospasm, arteriospasm, vasospasm, etc., with marked increase in blood pressure. The spasm may be localized but is more frequently diffuse, involving most of the arteries. The diastolic pressure assumes major importance and it may be found in the range of 110 to 120. Later, if the spastic contraction persists for any length of time, changes appear in the retina proper, such as localized areas of edema, to be followed still later by white cotton-wool patches and hemorrhages of various types. Varying degrees of vascular sclerosis usually appear. This picture represents further progression of the disease process and is of much graver prognostic significance.

This stage of the disease has been described

by Wagener and associates as "severe benign" type, since he has found that, although of a serious nature, many of these cases may improve with appropriate general treatment, and the retinal changes may also clear up in part. The hemorrhages and exudates absorb, and the spastic contraction in the arteries subsides. The patient is generally improved. Blood pressure, however, remains elevated, and similar recurrent episodes may develop with final progression of the disease until the characteristic picture known as "malignant hypertensive neuroretinopathy" appears.

THE retinal changes in this condition are quite characteristic. There is a swelling of the nerve head and surrounding retina due to edema, with numerous hemorrhages and white patches scattered over the fundus. The white spots are of two types—cotton-wool patches, and more sharply defined, glistening deposits, especially in the deeper layers of the retina and, at times, in the macular area, where they may assume the form of star-shaped striae or fan-like figures. The fluffy, soft, cotton-wool patches are produced in part by degeneration of the nerve fibers resulting from the ischemia which follows extreme vasospasm, recognized microscopically as cytoid bodies (Greear), and also by transudates of serum and precipitated fibrin in the deep retinal layers. The "hard" white spots and the white striae in the macula consist of masses of fat-laden cells, sometimes called bladder cells or "histocytes" (Greear). They are frequently referred to as "edema absorbing exudates" (Wagener).

Some authors claim this malignant phase of essential hypertension is, indeed, a separate clinical entity and, therefore, does not arise from a pre-benign process; other authorities state it may be the terminal phase of a long-standing benign type of hypertension. The disease is more common in the younger age group, from 30 to 50. Certainly such forms, as those seen in the toxemia of pregnancy, develop rapidly, and frequently there has been no antecedent hypertension or arteriosclerosis. I have frequently seen the disease in young people under 30.

The retinal vessels show marked constriction of the arteries and some branches may be reduced to mere threads. Frequently there is marked irregularity in the caliber of the vessels with alternate dilatation and constriction. Many of the finer vessels may be completely obscured by the intense retinal edema. The terminal branches of both arteries and veins are frequently tortuous. The blood pressure is usually very high, ranging from 200 to 290 systolic and from 130 to 160 diastolic, or higher. This fundus picture is of extremely grave significance, and few patients survive longer than a year or two after its appearance.

The various retinal changes described above may be conveniently classified as Grades I to IV, depending upon the extent and severity of the lesions encountered. In the absence of any general agreement concerning the classification of the sclerotic and spastic changes which appear in the vessels in hypertensive vascular retinal disease, the rough method of grading the degree of sclerosis I to IV, and spasm in stages I to IV has been found expedient.

The fundus changes in the toxemia of pregnancy and in some cases of chronic parenchymatous nephritis may be indistinguishable from those of malignant hypertension. It is now generally conceded that the severe hypertension which is also present in certain stages of these diseases is responsible for the retinal changes, so the retinal changes are the same for practical purposes though there may be some variations in the fundus picture recognizable by one expert in ophthalmoscopic interpretation. If there has been a pre-existing sclerosis of the retinal vessels of long standing, varying grades of this may be present and afford some information as to the duration of the disease process.

The writer has freely drawn upon various well known standard texts and atlases in the preparation of parts of this paper. The textbooks and atlases of C. Adam and of Axenfeld, and the more recent text of W. S. Duke-Elder have been found invaluable and are recommended to the student who wishes more complete information regarding this subject.

Roentgenologic Diagnosis of Diaphragmatic Hernia

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THE DIAGNOSIS of diaphragmatic hernia, broadly speaking, is essentially a roentgenologic problem. At the Mayo Clinic, 90 per cent of these hernias are found more or less unexpectedly at routine examinations of the stomach, or more rarely of the colon, to determine the cause of gastrointestinal symptoms. In the remaining 10 per cent of cases the patient comes with the diagnosis already made, or the clinical history is suggestive of hernia, or routine roentgenograms of the thorax reveal abnormalities that may be due to hernia and so call for further roentgenologic investigation.

In all roentgenologic examinations of the stomach the possible existence of diaphragmatic hernia should be kept in mind by the examiner, and his standard technic should provide for that contingency. Since a vast majority of hernias are at the esophageal hiatus, the region of the esophagogastric juncture should invariably be inspected thoroughly while the first two or three swallows of the barium suspension are being taken. Displacement or hooklike curvature or angulation of the lower portion of the esophagus is suggestive of hernia and is a common manifestation. Retardation of the barium stream at the hiatus, disparity between the upper level of the gastric contents and the site of the esophageal aperture, or what seems to be a high hourglass deformity should always make the examiner think of hernia. Any deviation from the normal size, contour, or general appearance of the lower part of the esophagus or any deviation from its normal relation with the stomach should cause the examiner strongly to suspect hernia and to demand further roentgenoscopic and roentgenographic study with the patient recumbent and strongly tensing his abdominal muscles. This technic is necessary to confirm the presence, and to demonstrate the extent, of herniation. Hernia in which only the colon or small bowel is implicated cannot be confirmed definitely or excluded confidently without employing the barium enema or observing the transit of the barium meal.

Roentgenologic manifestations of diaphragmatic hernia vary with the site of the lesion, the abdominal viscus extruded, and with the extent of extrusion. These factors in turn are related to other factors, such as the anatomy of the region involved and the cause of the hernia.

As might be expected, many classifications of hernia have been offered, almost as many as there are writers on the subject. Harrington[1] has proposed what seems to me to be the most complete and practicable classification of all,

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for it can be applied satisfactorily by the surgeon, the clinician, and the roentgenologist. Items in this classification that are of roentgenologic significance are as follows:

1. Hernia through the esophageal hiatus
   a. Congenitally short esophagus with thoracic stomach
   b. Esophageal hiatal hernia with shortened esophagus
   c. Esophageal hiatal hernia without shortening of the esophagus
   d. Para-esophageal hernia through the hiatus
2. Hernia through the foramen of Morgagni
3. Pleuroperitoneal hernia
4. Hernia through congenital defects in the diaphragm
5. Congenital absence of the hemidiaphragm
6. Posttraumatic hernia

HERNIAS THROUGH THE ESOPHAGEAL HIATUS

These constitute 98 per cent of the diaphragmatic hernias and are found at more than 1 per cent of all roentgenologic examinations of the stomach at the Mayo Clinic.

Congenitally short esophagus with thoracic stomach—This is a rare variety. In this type the esophagus is short and straight and the stomach is not truly herniated, since the supradiaphragmatic portion has never been below the diaphragm. In the single case seen at the clinic, five-sixths of the stomach was located above the diaphragm, and the hiatus was 12 cm. in diameter. The roentgenologic diagnosis of hernia should be self-evident in most cases; if the esophagus is extremely short it may fairly be assumed that the condition is congenital, but in less pronounced instances distinction from the next variety to be discussed is scarcely possible.

Esophageal hiatal hernia with shortened esophagus—This designation is intended to imply that originally the esophagus was of normal length but that it became shortened by tonic contraction following the hernia and that the term "thoracic stomach" does not apply. At operation the surgeon can base this diagnosis on the fact that the esophagus can be stretched to approximately its normal length. Of course the roentgenologist cannot determine whether such elasticity exists or not, but he will incline toward this tentative diagnosis because a large proportion of the hiatal hernias are of this variety, and the true short-esophagus thoracic-stomach variety is exceedingly rare.

Esophageal hiatal hernia without shortening of the esophagus—This term is chosen to designate those hernias in which the esophagogastric juncture is above the diaphragm, and the lower end of the esophagus is slightly or markedly redundant, a feature which distinguishes the group from the hernias with shortened esophagus. It is the most common type of hiatal hernia, and 66.5 per cent of the hiatal hernias observed roentgenologically at the clinic in 1944 were of this variety.

Para-esophageal hernia through the hiatus—This term designates a variety of hernia in which a portion of the stomach is extruded through the esophageal hiatus, but the esophagogastric junction remains below the diaphragm and the esophagus does not participate

in the hernia. Any part of the stomach may be involved, but most often the cardia is implicated. This group is small, constituting only 7.5 per cent of the hiatal hernias examined roentgenologically at the clinic in 1944.

IN EFFECTING the roentgenologic diagnosis of esophageal hiatal hernia and distinguishing among its varieties, certain items are of fundamental importance. First, the basic point of orientation in the diagnosis is the exact situation of the esophagogastric junction in relation to the diaphragm. Second, retardation of the barium stream at the hiatus is highly significant of hernia, for it occurs in almost all cases. Third, redundancy and tortuosity of the lower portion of the esophagus without dilatation is strongly suggestive of hernia, for it is present in more than one third of the cases.

Many technics have been devised for the demonstration of hiatal hernias. Satisfactory inspection of the esophagogastric juncture and its vicinity can usually be obtained in the customary anterior view with the patient standing. Then, as he takes the first swallows of the barium mixture, he should be required to strain his abdominal muscles in order to reproduce any hernia that may have been reduced. Pressure of the examiner's hand over the patient's stomach may also elicit such hernias. If these measures give negative results and doubt persists, roentgenograms should be made with the patient lying on his back and tensing his abdominal muscles.

HERNIA THROUGH THE FORAMEN OF MORGAGNI

This type of hernia, also termed "parasternal" or "costosternal" hernia, is quite uncommon. The so-called foramen is a bilateral, retrosternal, triangular space bounded in front by the sternum, medially by the sternal portion of the diaphragm, and laterally by the costal portion of the diaphragm. The hernia may be unilateral or bilateral, and either stomach or colon, or both, may be implicated. Occasionally only the omentum is involved. First evidence of the hernia is likely to consist of abnormal shadows near the midline and at the base of roentgenograms of the thorax. Lateral roentgenograms may reveal fluid and gas in the contents of the hernia, and such findings are strongly suggestive. Examination with the aid of the barium meal, or with the aid of the barium enema, since the colon is involved more often than the stomach, may establish the diagnosis.

PLEUROPERITONEAL HERNIA

This type of hernia, or hernia through the foramen of Bochdalek, is one located through a posterior segment of the diaphragm, a weak spot and point of fusion in the development of the organ. These hernias are congenital. Harrington has estimated that 75 per cent of infants with this condition die shortly after birth; few of the remainder reach adulthood. Bizarre shadows in the thorax are likely to be the first clue to the condition, and roentgenologic examination of the stomach, small bowel, and colon may lead to the diagnosis.

HERNIA THROUGH CONGENITAL DEFECTS IN THE DIAPHRAGM

These hernias, other than those through the foramen of Morgagni or Bochdalek, have been found most often in infants and children. Usually the hernia is through the left half of the diaphragm, and the stomach, colon, small bowel, or spleen may be implicated. Abnormal shadows at the base of the lung, especially on the left, in roentgenograms of the thorax are usually the first observed significant manifestations and call for examination of the alimentary canal with the aid of opaque media.

CONGENITAL ABSENCE OF THE HEMIDIAPHRAGM

Displacement of abdominal viscera into the thorax resulting from congenital absence of a hemidiaphragm is rare. Usually it is the left half of the diaphragm that is wanting. Roentgenograms of the thorax show striking abnormality of the affected side, and the presence of

the gas bubble or colonic haustra may indicate that the stomach or colon is in the thoracic cavity. However, administration of the barium meal or enema may be necessary to identify such displacement. In any case, the condition must be distinguished from other varieties of hernia and from eventration.

An exceedingly rare variety of congenital hernia is that resulting from congenital peritoneopericardial defect with herniation of abdominal viscera into the pericardial sac. A case has recently been reported by Wilson, Rumel, and Ross,[2] who have found only 8 similar cases recorded in the literature.

POSTTRAUMATIC HERNIA

Posttraumatic hernia may result from wounds by weapons or missiles, from crushing injury or violent forward flexion of the body, or from localized erosion or inflammatory necrosis of a segment of the diaphragm. Any portion of the diaphragm may be penetrated. Roentgenograms of the thorax constitute a common approach to the discovery and diagnosis of these hernias by disclosing abnormal shadows extending upward from the bases of the lungs. Occasionally the presence of the stomach or colon in the hernia is apparent, but complete and confident diagnosis requires examination with the aid of the opaque meal and enema. If only a solid viscus, such as the liver or the spleen, is implicated, diagnosis may be difficult.

COMMENT

Obviously, the roentgenologic diagnosis of diaphragmatic hernia depends on demonstrating the presence of part or all of an abdominal viscus above the diaphragm, or proving the existence of an aperture in the diaphragm through which an abdominal viscus can be extruded by an increase of the intra-abdominal pressure. In any case, the position of the diaphragm must be ascertained by tracing its curvilinear shadow, and this may be difficult in cases of gross traumatic rupture. In an overwhelming preponderance of cases the stomach or bowel participates in the hernia, and this fact can be established by administering a radiopaque meal or enema. In all such instances the differential diagnosis is evident. If, however, the hernial content consists of a solid viscus only, such as the spleen or omentum, the lesion is likely to be mistaken for a neoplasm at the base of the lung, though if the hernia is reducible the diagnosis should be evident. Distinction of several varieties of hernia from one another can usually be made. Many hernias will escape discovery unless the roentgenologist keeps the condition in mind at every examination of the thorax and of the alimentary canal.

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DIAGNOSTIC CLINIC

Treatment of Psoriasis

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Psoriasis is an acute, more often chronic, dermatosis originating as small, bright red papules, which rapidly or slowly increase in size to form raised, flat-topped disks or plaques of varying size and shape usually covered with dry, adherent scales. It has a predilection for the knees, elbows, and scalp, but the eruption may become generalized. At times the nails are involved. There is a seasonal variation with a majority of the exacerbations in the winter months. The general health is not usually affected except in those patients who have a complicating arthritis. The cause of the disease is unknown and treatment does not produce a permanent cure.

According to a statistical study,[1] psoriasis is one of the ten most common skin diseases, being eighth in incidence. It constitutes 6 per cent of all the skin diseases that are observed. It may occur at any age, the youngest reported case occurring in a baby six days old,[2] but ordinarily the disease begins in the second or third decade,[3] most often between the ages of 15 and 30 years. Uncommonly it first appears after the age of 45, but rarely past the age of 60. In a large series[4] of cases the ages of the patients varied from 6 to 82 years and the longest duration of the disease on record is fifty-five years.

Psoriasis occurs as frequently in females as in males and in the Caucasian race equally in blondes and brunettes, but the disease is rare in the Negro and is almost unheard of in the full-blooded Negro. In the latter a biopsy should confirm the clinical diagnosis of psoriasis and a blood serology should be obtained, because the eruption may be the psoriasiform type of secondary syphilis.

Psoriasis is noninfectious and noncontagious, but there is a familial incidence in 25 to 40 per cent of the cases. This is a familial predisposition and not a dominant characteristic.

The disease is common in the Arctic and Temperate zones, but occurs less often in the Tropical zone; in Panama, Central America and Southern Europe psoriasis is seldom observed. In the United States psoriasis is observed more often in Missouri than in Louisiana or Texas, whereas Chicago, Minneapolis, and Boston have a great deal more psoriasis than does St. Louis. Likewise in the Temperate zone the disease is more prevalent in winter than in summer. Approximately 75 per cent of

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the patients with psoriasis will develop recurrences or exacerbations of the eruption in cold weather, but will again improve with the onset of warmer weather in May and June. About 15 per cent claim there is no change with the seasons whereas 10 per cent state they are worse in the summer than in the winter.

The cause of psoriasis is unknown although numerous theories have been proposed. At present there are many advocates of the theory that a disturbed fat metabolism is the cause of the disease. Substantiating this opinion are the finding of a disturbed lipoid metabolism by Grütz and Bürger,[5] the observance of extracellular fat in the uninvolved skin of psoriasis patients by Madden,[6] the beneficial effect of sarsaparilla (which has an affinity for cholesterol) by Deneke[7] and Thurmon,[8] of lipotropic substances such as soybean lecithin by Goldman[9] and of lipocaic, a fat metabolizing pancreatic hormone by Stewart, Clark, Dragstedt and Becker.[10]

Becker[11] states that any factor resulting in mobilization of fats and lipoids from the tissues such as low fat intake, starvation, pregnancy and adrenal cortex will benefit psoriasis. Vitamins B, C, D and defatted wheat germ are essential to the metabolism of fats because they stimulate the function of the adrenal cortex.

Some investigators believe that a disturbed nervous mechanism causes psoriasis. Many psoriatics do have neurocirculatory instability and exacerbations have occurred following shock or prolonged nervous tension, indicating that the nervous system has an effect on the course of the disease. Trauma is a factor as evidenced by the presence of Köbner's phenomenon. This consists in the appearance of lesions of psoriasis in a scratch mark, but occurs only in the acute stage of the disease.

Others claim that psoriasis is an infection of mycotic, bacterial or virus origin. A type of rheumatoid arthritis, the so-called "psoriasis arthropatica," occurs in a small percentage of patients, usually after an acute exacerbation of a chronic psoriasis. It is thought to be due to absorption of toxic products into the system from the acute, disseminated lesions.

THE patient with psoriasis complains of few symptoms. In the acute outbreaks itching, smarting or burning may occur, but only a small percentage of patients in the chronic stages experience pruritus and then only of a mild degree. In thickened patches painful fissures may appear. The chief complaint is the embarrassment produced by the eruption, often curtailing activities such as athletic pursuits and pleasure-producing hobbies.

The earliest lesion of psoriasis is a pinhead sized papule of bright red color. The papules slowly or rapidly increase in size to form larger papules and nodules, which in turn coalesce to form plaques or patches. Some of the patches become quite large and may cover such a wide area as the entire surface of a leg or forearm. The earliest lesions are round or oval, but as they coalesce various shapes and forms are produced. By peripheral extension of the patches gyrate figures occur. Healing takes place by central clearing with a resultant ring form, having a paler, depressed midportion and elevated red margins. After the active lesion regresses there results a red or reddish-brown stain, which persists for a varying period of time and eventually disappears without scar formation.

The scale characteristic of the disease occurs in the earliest lesions and persists throughout. It is thick, silvery-white, mica or asbestos-like, adherent in the center and looser at the margins. When the scale is removed small bleeding points are revealed although they are not always visible to the naked eye.

The sites of predilection are the elbows, knees, the extensor surfaces of the forearms and legs, the scalp and the sacral region. The chest, abdomen, back, thighs and arms are frequently involved, but rarely the mid-face, fingers, toes and mucous membranes. In the acute stage and often in children the eruption is diffuse, but there may be a wide dissemination of the chronic lesions. Very infrequently, a pustular type of the disease occurs on the palms and soles. Most of the so-called cases of pustular psoriasis are in reality pustular bacterids as described by Andrews[12] or acrodermatitis perstans.

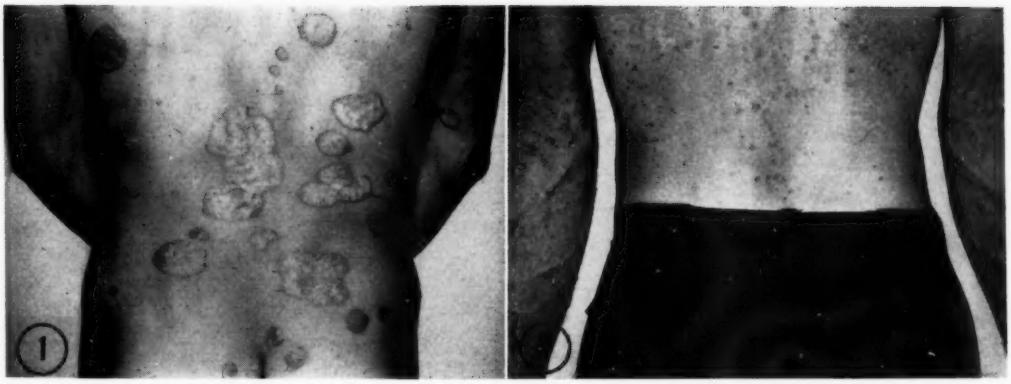

Figure 1. A chronic, discoid psoriasis. Plaques are covered with thick, adherent, silvery scales.

Figure 2. Scaly patches on the elbows, a common site for the lesions of psoriasis.

Figure 3. Extensive involvement of the anterior surfaces of the legs, another favorite site for the eruption. Scales have been removed by application of a keratolytic ointment.

Figure 4. Discrete patches of psoriasis in scalp margins.

Figure 5. Psoriasis nummularis of the trunk and intertriginous type of psoriasis in the axillae. Intertriginous type difficult to differentiate from seborrheic dermatitis.

Figure 6. Papular psoriasis of the abdomen and intertriginous psoriasis of the inframammary folds.

In chronic psoriasis there may be an involvement of finger and/or toenails in which the nails are of an opaque color with pitting, ridging and irregularity of the nail surfaces. The nail may separate at the base, may become raised from the nail bed by subungual scales and debris and finally may be destroyed.

An occasional complication is rheumatoid arthritis usually affecting the smaller joints of the hands and feet, the wrists and ankles, but also at times the larger elbow, knee, and shoulder joints. An odd occurrence in the experience of the author was the concomitant occurrence of rheumatoid arthritis in eight of the digits of the hands, which digits also revealed psoriasis of the nails, but the two digits without psoriatic nail involvement were also free of arthritis.

A disease which is difficult to differentiate from psoriasis is seborrheic dermatitis. Although both are prone to involve the scalp, the process is more uniform and diffuse in seborrhea. Likewise the eyebrows, mid-face and ears, sites where seborrhea most often occurs, are rarely involved by psoriasis. The color of the seborrheic eruption is yellowish-red as compared to the bright red of psoriasis and the scales are more loosely attached and greasier in seborrhea. In many cases it is difficult and at times impossible to differentiate between these two eruptions, particularly if the axillary, inframammary and crural regions are involved.

Pityriasis rosea involves chiefly the trunk, arms and thighs and rarely appears on the scalp or legs. The eruption is more acute, more evanescent, more pruritic than that of psoriasis and the scales are thinner. Typically the long axes of the macules of pityriasis rosea parallel the cleavage lines of the skin.

The secondary eruption of early syphilis, particularly if it occurs in the Negro, may cause confusion in diagnosis. In the Caucasians the color of the secondary rash of syphilis is of a coppery-red shade as compared to the bright red of psoriasis. Also the scale is not as thick or adherent as in psoriasis and tends to form a collarette at the periphery of the lesion. In both whites and Negroes the eruption of syphilis is arranged in circles or segments of circles in conformity to the vascular supply of the skin, while the palms, soles and face which are not frequently affected in psoriasis are often involved. Mucous membrane lesions, moist papules around mucocutaneous orifices, the general symptoms of headache, malaise, fever, the history of an initial lesion and the positive serology are all aids in differentiating syphilis from psoriasis.

Ringworm infection of the skin usually is manifested by macules or plaques with clearer, slightly depressed central portions and raised red margins. Such lesions resemble a regressing psoriasis, which tends to fade first in the central portion. The scale of a tinea eruption is less thick than that of psoriasis and the fungus can be demonstrated by direct examination of the scales or can be grown in a suitable culture medium. Ringworm infection of the scalp produces alopecia with broken hairs, a phenomenon which does not occur in psoriasis.

Nummular eczema of the extremities is more vesicular, more moist, more crusted and more pruritic than psoriasis.

The treatment of psoriasis cannot be successful unless the physician obtains the complete cooperation of the patient. An honest explanation should be made that although the present eruption can probably be eradicated, a promise of permanent cure cannot be made. Encouragement should be given by stating that the disease is not infectious, not contagious and almost never becomes malignant.

Many diets have been prescribed but all have been ineffective except the low fat diet, which has proved beneficial in some instances. Vitamins B, C, D and defatted wheat germ are of value because they are essential to the metabolism of fats. Sarsaparilla, which supposedly neutralizes the increased amount of cholesterol in the psoriatic is usually prescribed in the form of sas par tablets. They are well tolerated and are of value at times.

The drug which has been most effective in psoriasis is arsenic, administered as Fowler's

Figure 7. Psoriasis gyrata et figurata.

Figure 8. A rare manifestation of psoriasis on the mucous membrane of the penis. The red patches of psoriasis on the inner thighs resemble tinea cruris.

Figure 9. Psoriasis of the fingers, an area in which the disease seldom occurs.

Figure 10. Psoriasis of the nails with psoriasis arthropatica of the right third finger.

Figure 11. Pustular psoriasis of the palms.

solution (liquor potassi arsenitis). The daily dosage ranges from 6 drops at the onset with gradual increase to a maximum of 24 to 30 drops, normally given in divided doses. Symptoms of intolerance to the drug, such as eyelid edema, gastrointestinal cramps and diarrhea, necessitate a temporary discontinuance or a reduction in dosage. Arsenic should not be continued longer than ten or twelve weeks without a rest period because of its tendency to produce keratoses and carcinomas of the skin and its capability of inducing an exfoliative dermatitis. Furthermore, in a period of two to three months the success or failure of arsenical treatment can be determined. Arsenic trioxide in a dosage of 1/32 to 1/16 gr. three times daily, usually given as the Asiatic Pill is almost as efficient as Fowler's solution and often is better tolerated. Patients and pharmacists should be warned that prescriptions containing arsenic should not be refilled without the approval of the prescribing physician.

Autohemotherapy, which consists of the withdrawal of blood from the vein of the patient and immediate reinjection into the buttocks has been helpful on some occasions.

The sulfonamide drugs, penicillin, and streptomycin are of no value in psoriasis. The salicylates may give relief from the pain of arthropathic psoriasis, but oddly the arthritis improves as the lesions of psoriasis fade and the arthritis becomes worse during an exacerbation of the eruption.

In the local management of the disease, bathing, which is often contraindicated in inflammatory skin eruptions, is of value because it removes the scales and thus permits the prescribed medicament to come in more intimate contact with the surface of the lesion.

Sunlight is an efficient therapeutic agent. Most patients with psoriasis obtain more relief in summer than in the winter and those areas exposed to the sun clear more rapidly. When sunlight is not available the ultraviolet light or the cold quartz light may be substituted although they are much less effective. However, sunlight and ultraviolet light will often make an acute psoriasis worse and exposure to them should be avoided in the acute stages of the disease.

Liquor carbonis detergens, a mixture of coal tar and soap bark tincture, is the most valuable topical agent. Applied in full strength once or twice daily, it may be combined with exposure to sunlight or ultraviolet light, as tar, a photosensitizer, enhances the effect of these rays. Most patients tolerate the applications well and the rapidly drying solution is more pleasant to use than an ointment.

Chrysarobin, the standby of dermatologists for years, has the disadvantages of staining the skin, clothing, and bedding a brown color and of producing a severe conjunctivitis. This precludes its use on psoriasis of the scalp and face. Dihydroxyanthranol with the trade name of anthralin is a good substitute for chrysarobin. It is applied as an ointment varying in strength from 0.25 per cent to 2 per cent, does not stain as deeply or permanently as chrysarobin and can be applied to the scalp.

Other ointments which are helpful are those containing salicylic acid in 5 to 10 per cent strengths combined with ammoniated mercury or sulphur. These are particularly efficient in those eruptions which resemble seborrheic dermatitis.

Many patients, particularly women, object to the use of ointments on the scalp. For them the following lotion may be beneficial:

Rx	Paraffin oil	140.0
	Oleic acid	70.0
	Oil of lavender	1.2
	Phenol	1.2
	½ of 1% sodium chloride water q.s.	240.0

It also can be used on the glabrous skin, but occasionally will produce a folliculitis which may be more irritating than the psoriasis. If folliculitis occurs the following lotion may be substituted:

Rx Liquid petroxylin (N.F.) . . . 120.0
Phenol 1.2
Sodium chloride 1.2
Oil of lavender 0.2
Aqua destillata q.s. 240.0

For solitary patches which have been resistant to all treatment, x-ray therapy may be used. However, the number of previous roentgen exposures and the total dosage should be closely checked, because psoriasis will recur just as readily after x-ray treatment as after any other type of therapy. An individual is far better off with psoriasis alone than with psoriasis plus a severe radiation reaction.

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DAS NARRENSCHNEIDEN
By Pieter Brueghel (1556)

CASE REPORT

Congestive Splenomegaly With Cytopenia

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Congestive splenomegaly with cytopenia has such varied clinical and laboratory manifestations that each case is worthy of study. In the case to be presented, all findings are in keeping with the above diagnosis and yet do not include all symptoms or signs listed in previously reported cases or in textbook descriptions of this illness. The response to splenectomy in this case was dramatic and encouraging in that a follow-up study at the end of a six-month period revealed sustained subjective and objective improvement.

CASE PRESENTATION

A 50-year-old white male was admitted to Deshon Veterans Administration Hospital in February 1947. At the time of admission the patient's complaints were diarrhea, pressure and pain in the abdomen, loss of weight, and extreme fatigue.

The patient was a veteran of World War I. Family history was negative except for diabetes in a sister and a cerebral accident as the cause of the father's death. This man was first hospitalized in 1922 for pleurisy and severe cough. A diagnosis of tuberculosis was made, and from that time forward there were numerous hospitalizations for pneumonia and recurrent pulmonary hemorrhages. Approximately four years before admission this patient was hospitalized for pneumonia. During this latter hospitalization "a lump" was discovered in the abdomen. After complete x-ray studies, surgery for excision of "the lump" was recommended. The patient was not in favor of this procedure and so left the hospital against medical advice. In 1944 the patient was again hospitalized for pneumonia and since that time has been unwell.

The patient suffered from severe cough, weight loss, weakness and fatigue, and dyspnea on exertion. There was no history of ankle edema, orthopnea, or precordial pain. During the nine-month period before admission, the patient had eight to twelve loose stools daily. The stools were brownish in color and stringy in character. There was no history of melena or hematemesis. There were no periods of constipation. During this time the patient had a sensation of pressure and fullness in the upper abdomen which was aggravated by bending. Since November 1946 the abdomen had become swollen and the "lump" in the abdomen had become progressively enlarged. Because of the above signs and symptoms, hospitalization was recommended.

Physical examination revealed a bald-headed,

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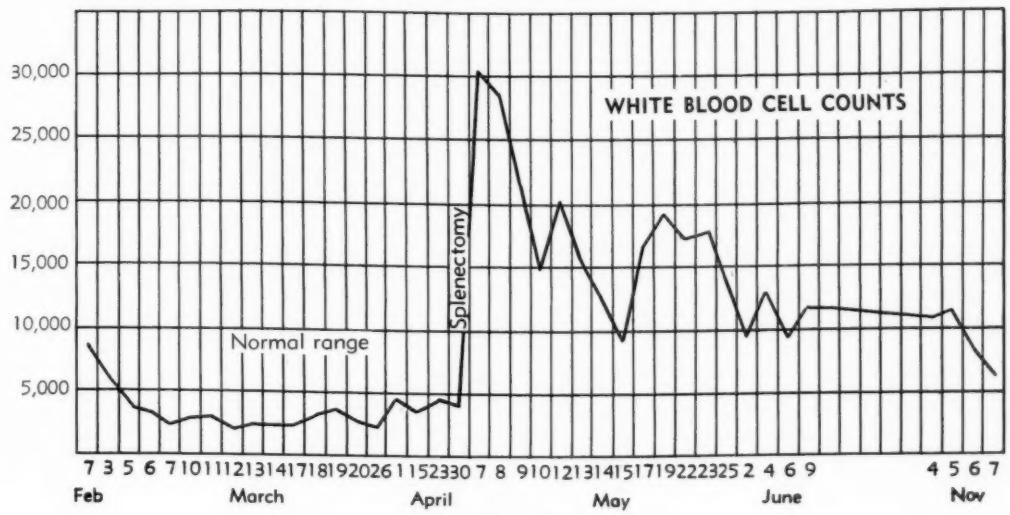


Graph 1

white male of fifty years in no acute distress. Examination was essentially negative except for numerous coarse râles throughout the chest bilaterally. Râles were not constant in location and disappeared after diagnostic cough. Examination of the abdomen revealed two large masses. One was in the upper left quadrant and was free and movable and extended to a point below the level of the umbilicus and almost to the midline. The mass seemed notched at its inferior border. The other palpable mass was in the upper right quadrant and was rather rectangular in shape and did not extend to the right flank, but was palpable near the midline of the abdomen and extended for four finger-breadths below the right costal margin.

Because of the chest findings and history of previously active tuberculosis, the patient was studied to rule out active pulmonary tuberculosis. Six sputum examinations were negative for acid-fast bacilli, the patient was afebrile, there was a normal sedimentation rate and the chest x-ray was negative. After active pulmonary tuberculosis was ruled out, the patient was studied to determine the cause of his abdominal trouble.

Further questioning revealed that while the patient was hospitalized for pneumonia four years previously, he was treated with sulfadiazine and it was found that he had a leukopenia and therapy was discontinued. After a lapse of ten days, however, the sulfa therapy was reinstated. Thus, the

TABLE 1

BONE MARROW SMEAR, APRIL 17, 1947

Type of Cells	Per Cent Found	Per Cent Normal
Myelocytes	10.0	10-20
Neutrophils	9.0	
Eosinophils	1.0	
Juveniles	19.0	20-25
Stabs	17.0	5-10
Segmented	37.0	22.0
Neutrophils	34.0	20.0
Eosinophils	3.0	2.0
Lymphocytes	13.0	10-20
Monocytes	1.0	1-5
Megaloblasts	0.0	2.0
Erythroblasts	1.0	2-4
Normoblasts	6.0	22-35

Summary: Myeloid-erythroid ratio—13:1. No megakaryocytes seen. Depression of erythropoiesis.

Hematologic diagnosis: Hypoplastic anemia.

With the leukopenia and thrombocytopenia in this case, along with the anemia and presence of esophageal varices and a large mass in splenic region, we believe that this patient has Banti's disease. We feel that he is in the stage of the disease where the cirrhosis of the liver is far advanced and that he has been fortunate in not having any bleeding as yet, but bleeding may occur at any time. Suggest laparotomy with removal of the large mass which we feel is a Banti's spleen. (G. J. Brilmyer, M.D., pathologist.)

Figure 1. This roentgenogram shows depression and compression of the left kidney with kinking of the left ureter caused by the enlarged spleen.

first incidence of leukopenia occurred four years before admission. Frequent white counts were ordered after this information was obtained. (See Graph 1.)

Various x-ray studies were carried out:

1. A gastrointestinal series revealed that the stomach was displaced to the right. Also esophageal varicosities were discovered on fluoroscopy.

2. A barium enema showed that the splenic flexure of the colon was displaced to the right. The colon was normal.

3. An intravenous pyelogram showed that the left kidney was compressed and depressed by a mass in the upper left quadrant. The left ureter was kinked.

4. Retrograde pyelogram confirmed findings of the intravenous pyelogram. (See Figure 1.)

Various liver function tests were done and yielded results that were within normal limits. (See Table 2.)

Because the platelet count was below normal and because there was a persistent leukopenia and normal differential count, a diagnosis of congestive splenomegaly with cytopenia was made. This diagnosis was made despite the absence of a severe anemia. (See Graph 2.) Bone marrow

TABLE 2

LIVER FUNCTION TESTS

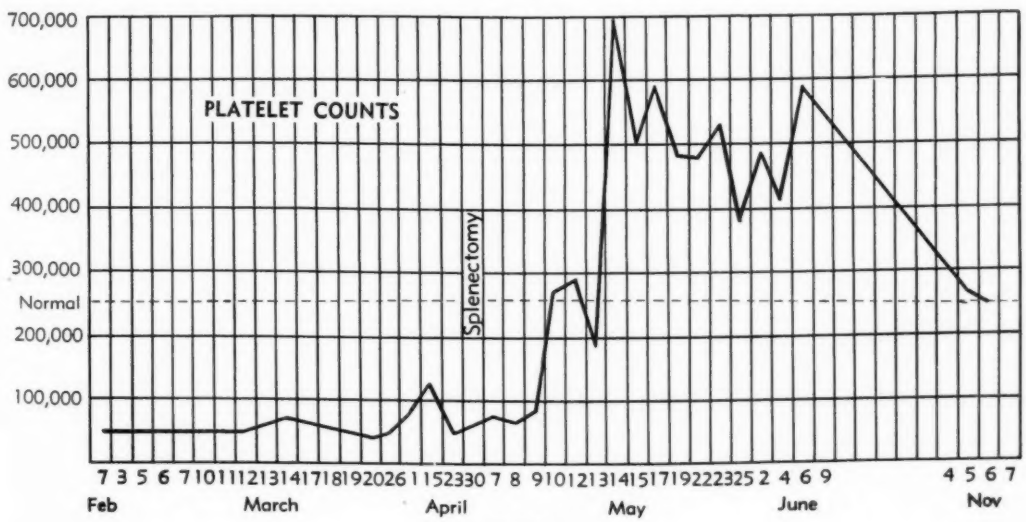
	Preoperative	Postoperative
Total protein and A/G ratio		
Total protein (in gm.)	7.16	7.52
Albumin (in gm.)	4.37	4.22
Globulin (in gm.)	2.79	3.30
A/G ratio	1.6/1	1.1/1
Cephalin Flocculation test		
24 hours	1 plus	2 plus
48 hours	1 plus	3 plus
Bromsulfalein (5 mgm. per kilo of body weight)	6.3% recovery in 45 minutes	11% recovery in 45 minutes
Serum bilirubin (mgm.)	0.28	0.78
Prothrombin time (per cent)	100	100
Ratio of cholesterol esters (per cent)	41	75
Icteric index	6 units	8 units

smear (sternal puncture) revealed depression of erythropoiesis with a myeloid-erythroid ratio of 13:1. (See Table 1.)

Surgical consultation was requested, and splenectomy was recommended. On May 6, 1947, an abdominal exploration and splenectomy was performed by one of us (L. E. B.). Upon opening the abdomen, a large spleen was visualized, adherent on all its surfaces to diaphragm, stomach, and pancreas. The spleen was freed from its numerous adhesions and after delivery of the spleen, the splenic pedicle was visualized. The vessels of the pedicle were extremely large and veins tortuous. Palpation of the liver by the surgeon revealed no gross hepatic pathology, but the left lobe was slightly enlarged. There were no signs of stenosis of the splenic vein or of other obstructive phenomena in the portal system. The patient made an uneventful postoperative recovery, and laboratory studies made postoperatively revealed some interesting findings. (See Table 2 and Graphs 1, 2, and 3.)

PATHOLOGIC REPORT ON THE SPLEEN

Gross—(See Figure 2.) The specimen shown here consists of a spleen which weighs 1,250 grams and measures 20 by 14 by 9.5 cm. The capsule in general is slightly roughened by scattered, shredded thin adhesions and on the lateral surface shows two dime-sized areas of thickening of the zuckerguss type. The consistency is moderately firm and rubbery. The cut surfaces of the organ reveal a thin capsule and a smooth, firm, beefy red pulp with indistinguishable malpighian corpuscles. All the cut surfaces are irregularly and relatively



Graph 2

Figure 2. Cross section of spleen showing cut surfaces with thin capsule, smooth beefy red pulp, and areas of fibrosis.

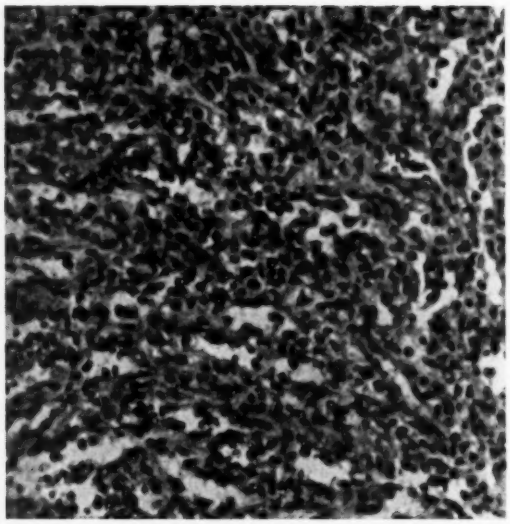


Figure 3. High powered microscopic section showing sinusoids thickened and rigid.

sparsely stippled by tiny purplish-black areas, in the center of which there are often siderotic nodules—an opaque brownish yellow material of rather tough consistency. These combined areas rarely exceed 2 mm. in diameter. The larger vessels are not grossly remarkable.

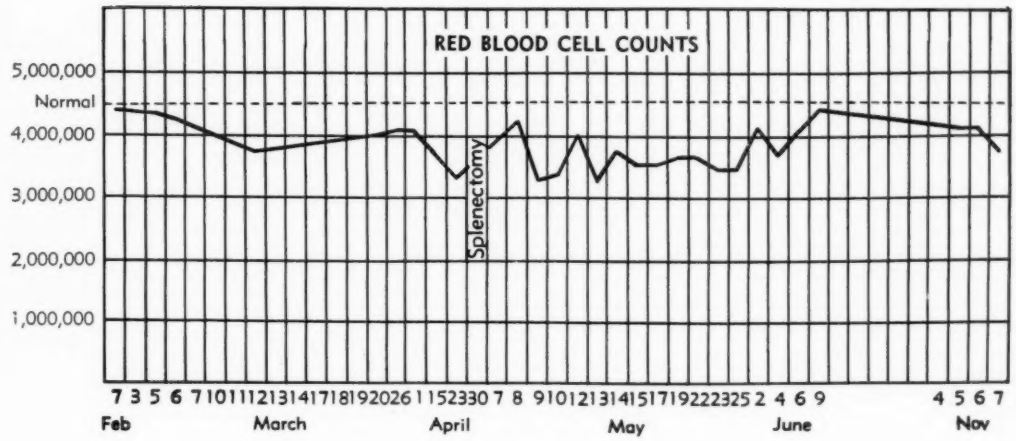
Microscopic—(See Figure 3.) Sections examined reveal a generalized subcapsular fibrosis with hemorrhage, although the capsule and trabeculae are not notably thickened. Numerical and dimensional decrease of the malpighian corpuscles is prominent, as are periarterial and perifollicular hemorrhages, both old and recent. The sinusoids are narrow and their walls are thick and "rigid." Phagocytosis is not unusually conspicuous.

DISCUSSION

This case is presented because of several unusual features. First, tremendous splenomegaly with the depression of two of the formed elements of the blood. It has been shown that anatomic enlargement of the spleen may be accompanied by depression of all the formed elements of the blood singly or collectively. Normally, the spleen is an organ in which blood cell formation, blood cell sequestration, and blood cell destruction are balanced physiologic functions. Any disturbance of this balance may lead to a variety of clinical syndromes. These disturbances may be primary (congenital) or secondary (acquired). For functional and pathologic consideration, the spleen, according to Doan and Starr Wright[1] is divided into (1) vascular system, (2) lymphoid system, and (3) reticulo-endothelial system.

When there is hyperplasia of the highly phagocytic cells, there follows destruction of red blood cells (hemolytic icterus), destruction of platelets (thrombocytopenia), and destruction of granulocytes (splenic neutropenia). These conditions are definitely relieved by splenectomy, which corrects the panhematopenia due to splenic instability.

Second, the cytopenia produced no untoward hemorrhagic effects. Symptoms which prompted



Graph 3

medical attention were those due to mechanical pressure by the spleen on the abdominal viscera. Third, following splenectomy, there was marked improvement in the subjective symptoms and laboratory findings. (See Graphs 1, 2, and 3.)

THE cause of this patient's splenomegaly is not clear. As early as 1904, Dock and Warthin[2] believed that the histologic changes in spleens of "Banti's syndrome" could be explained by prolonged passive congestion. These histologic changes were similar to those found in the case we are presenting. Larrabee[3] in 1934, after a study of 47 cases, felt that the condition was dependent upon various intra-abdominal lesions which produced obstruction to outflow of blood from the spleen.

In 1933, Turley[4] demonstrated that phagocytosis of perhaps extracellular enzymatic destruction of polys occurs in the spleen. He suggested that one of the functions of the spleen was disposal of polymorphonuclear cells in a normal individual. It was not until 1939 that Wiseman and Doan[5] suggested that neutropenia was due to increased phagocytosis of neutrophils by splenic clasmatocytes. In several cases under study by these authors such manifestations as thrombocytopenia and hemolytic anemia were present, and they suggested that the spleen had undergone a change in activity so that not only formed elements which had outlived their life span were destroyed, but also mature functional elements were destroyed.

Whipple[6] and his associates have found that the relatively great increase in splenic vein pressure in cases presenting "Banti's syndrome," when compared with venous pressures taken simultaneously on the arm, suggest that portal hypertension may be an important factor in the production of chronic splenomegaly. It is Whipple's conception that the so-called "Banti syndrome" is the result of mechanical obstruction to the flow of blood within the portal system which leads to increased portal pressure causing an engorgement of the spleen with subsequent splenomegaly. The obstructive lesion in the portal bed may be intrahepatic (cirrhosis) or extrahepatic (traumatic or inflammatory scarring and stenosis of the portal veins, cavernoma of the portal vein, stenosis of the splenic vein).

The diagnosis of the site of portal block may be suggested by several liver function tests such as high bromsulfalein retention, positive hippuric acid test, reversal of A/G ratio, and cephalin flocculation test positive. These findings would indicate intrahepatic block. If the above-mentioned tests were negative, we could assume that an extra-

hepatic block was present. Using the above criteria as a basis, it is then reasonable to assume that the patient we presented had extrahepatic block.

Whipple states that the site of portal obstruction is difficult to locate, even at operation, and that the site of portal bed block was not demonstrable in over 50 per cent of cases at the time of splenectomy at the Spleen Clinic at Presbyterian Hospital in New York. At recent operations done at the Spleen Clinic, diodrast venograms made at the time of determination of portal vein pressures, with roentgenograms at the operating table, have located the site of obstruction more frequently.

Splenectomy in the case we presented may have been curative. If the site of obstruction in the portal system was in the splenic vein, permanent cure will result. If the block was in the portal vein, relief will have been provided for a variable period of time because of the removal of a large area of the portal bed and the interval required for portal hypertension to be built up again.

The patient returned for study six months postoperatively. Hematologic studies showed the cytopenic improvement following splenectomy was sustained six months later. Studies of liver function showed very little change from previous studies done following splenectomy. Clinically, the patient is well and symptom-free. Thus far, the benefits achieved by splenectomy have continued for a period of six months and it is felt that in this case splenectomy was warranted and of definite therapeutic value.

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GENERAL PRACTICE RESIDENCY SET FOR NAVAL HOSPITALS

Recognizing the great need for the general practitioner in the field of medicine, the Professional Division of the Bureau of Medicine and Surgery of the U.S. Navy has inaugurated a new residency in general practice in Naval hospitals.

The program, as announced by Rear Admiral Clifford A. Swanson, MC, USN, Surgeon General and Chief of the Bureau of Medicine and Surgery, will cover a period of three years and will include that phase of medicine which is required to augment the several medical specialties recognized by the American Specialty Boards. The course has been designed to supplement the residency training program, now in effect, which is designed toward specialization of physicians.

During each year of the residency medical officers will receive six months of training in general medicine and related specialties, and six months in general surgery and related specialties.

Staffs of the Naval hospitals and outstanding civilian visiting staffs will serve as instructors for the training program.

SCIENTIFIC EXHIBIT

The Two-Hour Test for the Early Diagnosis of Pregnancy

HERBERT KUPPERMAN, Ph. D., M. D. and ROBERT B. GREENBLATT, M. D.

NEW YORK UNIVERSITY COLLEGE OF MEDICINE, NEW YORK CITY, NEWARK CLINICAL GROUP, NEWARK, AND UNIVERSITY OF GEORGIA SCHOOL OF MEDICINE, AUGUSTA.

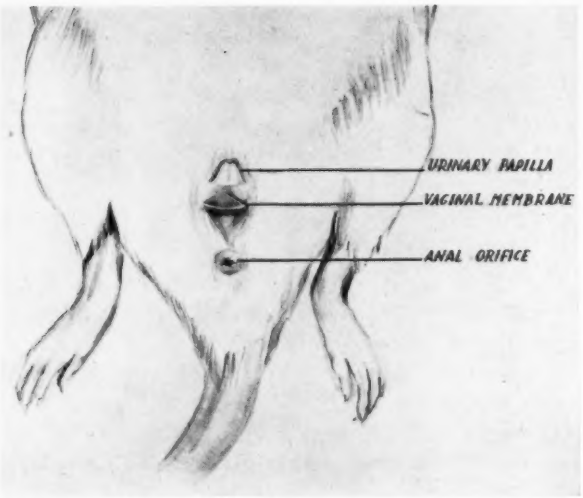
Following the advent of the classical A-Z pregnancy test, numerous attempts have been made to shorten the original procedure. The method utilizing the action of pregnancy urine in producing ovarian hyperemia, first described by Eberson and Silverberg in 1931, yielded the most rapid results. The evolution of tests dependent on this diagnostic endpoint is depicted below.

EVOLUTION OF THE TWO-HOUR PREGNANCY TEST EMPLOYING OVARIAN HYPEREMIA AS ITS DIAGNOSTIC ENDPOINT

Author	Year	Time Required for Completion of Test
Aschheim-Zondek	1928	96 hours
Eberson-Silverberg	1931	36 hours
Reiprich	1933	30 hours
Walker-Walker	1938	30 hours
Kelso	1940	24 hours
Frank-Berman	1941	24 hours
Salmon-Geist	1942	6 hours
Kupperman-Greenblatt	1943	2 hours

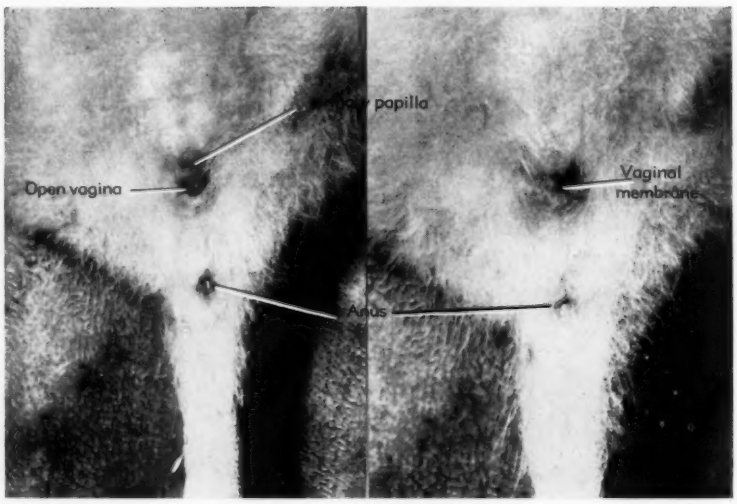
TECHNIC OF THE TWO-HOUR RAT PREGNANCY TEST

Selection of rats suitable for use in the two-hour pregnancy test depends on the state of the vaginal membrane. This membrane is intact in the immature animal but becomes patent when the rat attains sexual maturity. Since only immature rats are satisfactory for use in the two-hour procedure, only those animals exhibiting an intact vaginal membrane are used.



Left. Diagrammatic presentation of the external genitalia of the immature female rat.

Below. Photographs of external genitalia of the female rat.



Right. Patent vagina—evidence of ovarian activity. This animal cannot be used for the two-hour test.

Extreme right. Vaginal membrane intact—evidence of sexual immaturity. This animal may be used for the two-hour pregnancy test.

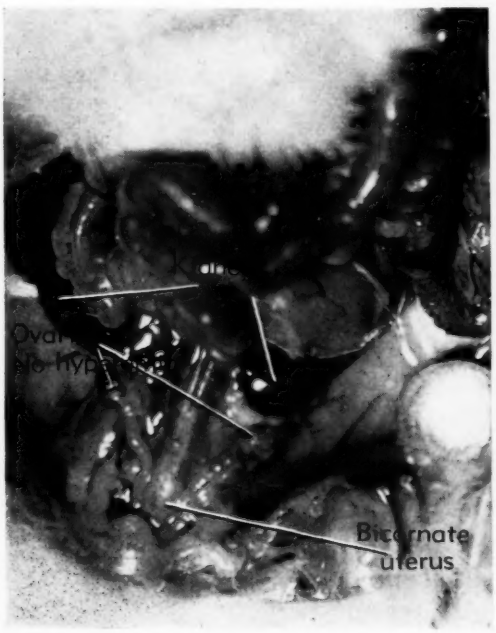
METHOD OF ADMINISTRATION OF URINE

ETHER ASPHYXIATION

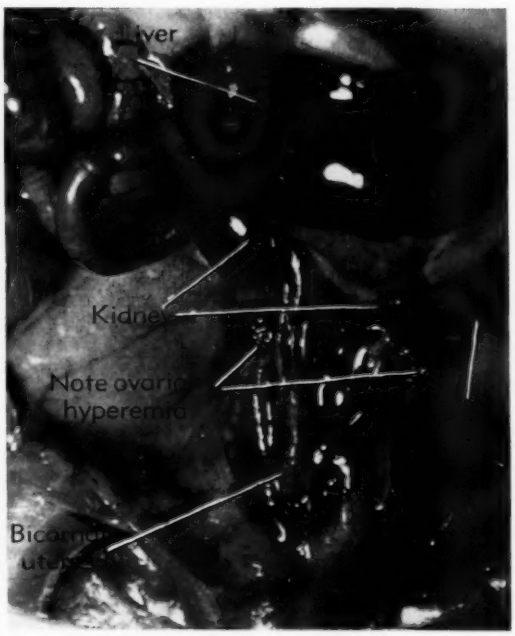
Left. The animal is held with its head in a dependent position, thereby permitting the viscera to gravitate toward the head, thus entailing little if any injury to the gastrointestinal tract by the intraperitoneal injection. One cubic centimeter of urine is injected intraperitoneally into both the right and left lower abdominal quadrants.

Right. It is important that asphyxiation be accomplished with ether since the use of other agents such as illuminating gas, etc., may predispose to false positive readings.

VISCERA EXPOSED

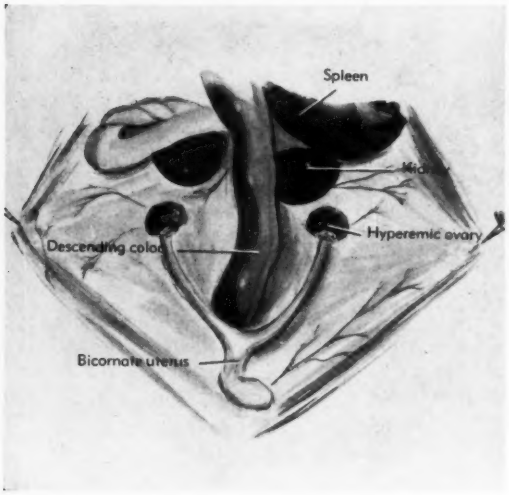


Negative rat

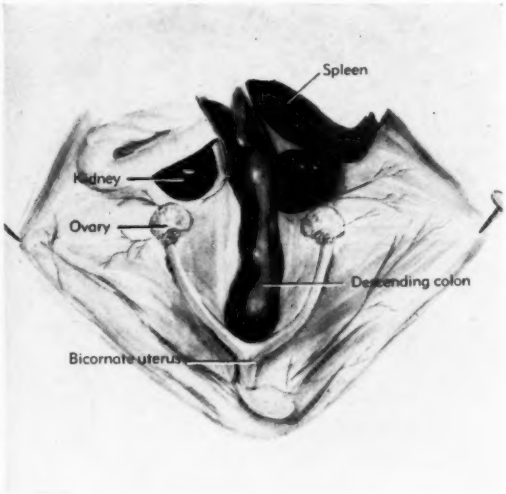


Positive rat

Pictorial sketch of ovaries, oviduct and uterus of negative and positive tests in rats injected with normal and pregnancy urine respectively.



Positive. Note ovarian hyperemia in contrast to lack of hyperemia of the oviduct and uterine horns.



Negative. Note oviduct and ovarian capsule are similar in color.

PRECAUTIONS TO BE NOTED IN PERFORMING THE TWO-HOUR PREGNANCY TEST

1. The animals, before examination, must be killed by ether asphyxiation.
2. In the process of exposing the ovaries, no loss of blood should result.
3. Examination of the ovaries should be made with as little trauma as possible to the surrounding tissues.
4. Ovarian hyperemia should not be read under fluorescent lighting or sunlight.
5. The hyperemic reaction is intensified if the ovaries are examined at least one to two minutes after exposure.
6. Positive reading may not be expected unless urine is examined at least one week to ten days after the missed menstrual period or twenty-one to thirty-five days after conception.

THE DIAGNOSTIC ACCURACY OF THE TWO-HOUR PREGNANCY TEST*

	Number of Tests	Errors	Per Cent Accuracy	Number of Rats
Positive	641	1	99.8	1148
Negative	636	5	99.2	1242
Total number	1277	6	99.5	2390

**Not including established cases of ectopic pregnancy*

RESULTS OBTAINED WITH THE TWO-HOUR PREGNANCY TEST IN ESTABLISHED AND SUSPECTED CASES OF ECTOPIC PREGNANCY

	Number of Cases	Correct Diagnoses	Incorrect Diagnoses	Per Cent Accuracy
Established cases of ectopic pregnancy	31	26	5*	83.9
Suspected cases of ectopic pregnancy	30	30	0	100
Total	61	56	5	91.8

**Incorrect diagnosis: 3 cases necrobiosis of villi; 2 cases—few viable villi*

The two-hour test, by reason of its rapidity and accuracy, attains its prime purpose in its utilization in the detection of ectopic pregnancy. Ectopic pregnancy, an obstetrical emergency, requires rapid operative intervention as soon as the diagnosis is made. In an emergency of this type the more lengthy tests cannot be employed. The value of the two-hour test in confirming the diagnosis of ectopic pregnancy is indicated in the above chart.

COMPARISON OF THE ACCURACY OF THE TWO-HOUR TEST WITH THAT OF THE FRIEDMAN TEST

	Test Employed	Number of Tests	Errors	Per Cent Accuracy
Positive readings	Two-hour test	163	0	100
	Friedman test	155	6	96.1
Negative readings	Two-hour test	155	0	100
	Friedman test	163	4	97.5
Total number tests	Two-hour test	318	0	100
	Friedman test	318	10	96.9

Five of the six false positives noted with the Friedman test were from urine specimens of menopausal patients. Since menopausal urine is rich in FSH activity, there is sufficient gonad-stimulating activity to produce ovarian stimulation and corpora lutea in the rabbit. However, FSH would not be expected to give a positive test with the two-hour procedure since LH is necessary to produce ovarian hyperemia (see chart, p. 524). Consequently menopausal urine, while capable of producing a positive reading in the rabbit, will fail to cause a false positive reaction when the two-hour procedure is employed.

GONADOTROPHIC HORMONES AND OVARIAN HYPEREMIA IN THE RAT

Extract	Dose[1]	Hyperemic Response
Follicle stimulating hormone (FSH)	1 gram	Negative
	2 grams	Negative
Luteinizing hormone (LH)	0.5 gram	Positive
	1.0 gram	Positive
Lactogenic hormone I	0.5 gram	Positive (weak)
	1.0 gram	Positive
Lactogenic hormone II	1.0 gram	Positive
Unfractionated gonadotrophic extract (sheep)	50 mgm.	Positive
	100 mgm.	Positive
Horse pituitary suspension	5 mgm.	Negative
Thyrotropic hormone (Armour)	10 units[2]	Negative
Purified pregnancy mares' serum (PMS)	0.1 mgm.	Positive
	0.25 mgm.	Positive
Purified pregnancy urine	3 cc.[3]	Positive
	4 cc.	Positive

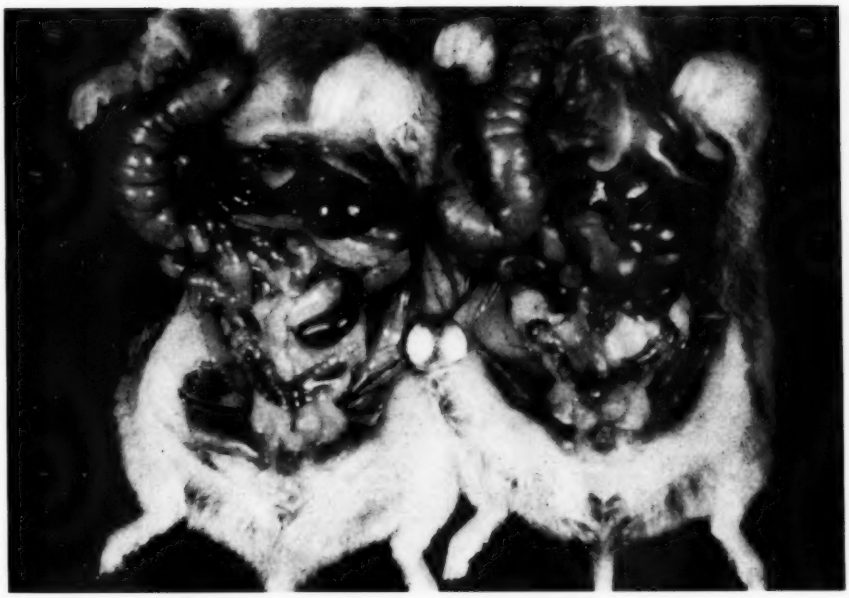
1. Doses of pituitary preparations expressed in equivalents of dried pituitary glands
2. Rowland-Parkes' units
3. Equivalent of unconcentrated urine

The results presented on the effect of various gonadotrophic extracts in producing ovarian hyperemia show that gonadotrophins containing only luteinizing or luteotrophic activity were effective in inducing hyperemia of the ovary. Thyrotrophic and FSH preparations in the doses employed were ineffective in causing ovarian hyperemia.

ADVANTAGES OF THE TWO-HOUR TEST

1. It is the most rapid accurate biological test proposed to date.
2. Excluding cases of ectopic pregnancy an accuracy of 99.5 per cent has been attained.
3. The test animals are readily procurable and considerable variation with respect to age and weight is permitted in the choice of the animal.
4. There is no difficulty in administering the urine.
5. The actual performance of the test requires only two to three minutes of the operator's time.
6. The endpoint is easily read and requires little experience to attain a high degree of accuracy.
7. Performance of the test does not subject the patient to any discomfort or inconvenience.
8. There is no need to purify, concentrate or alter the *p*H of the urine prior to injection.
9. Although a morning specimen is desirable, it is not essential.

Negative *Positive*
Ovarian hyperemia in the mature albino mouse.



Negative *Positive*
Ovarian hyperemia in the immature golden hamster.

THE RESULTS OF HYPEREMIC TEST IN ADULT MICE AND IMMATURE HAMSTERS*

	Positive Readings			Negative Readings		
	Number of Patients	Correct Diagnoses	False Positives	Number of Patients	Correct Diagnoses	False Negatives
Adult mice	103	98	5	62	59	3
Immature hamsters	69	65	4	49	47	2

**Animals sacrificed 15 hours after intraperitoneal administration of urine*

While ovarian hyperemia may be induced in immature hamsters and in adult mice by the urine of pregnant women, fifteen hours were required for the completion of the test and an overall accuracy of 95 to 98 per cent was attained. At least 3 mice or hamsters must be used for each test since a high percentage of the animals are poor reactors. Immature mice and guinea pigs were not satisfactory for use.

COMPARISON AND EVALUATION of the two-hour test with three of the procedures most commonly employed in the laboratory diagnosis of pregnancy.

TWO-HOUR RAT TEST

Animal	Immature female rat 21-55 days of age, 30-80 gm. in weight.
Urine	A total dose of 2 cc. of unconcentrated urine (morning specimen is not necessary) is injected intraperitoneally into the lower right and left abdominal quadrants.
Time	Two hours are required for the completion of the test.
Endpoint	Ovarian hyperemia.
Accuracy	99.5 per cent.

FRIEDMAN RABBIT TEST

Animal	Isolated adult female rabbit.
Urine	10 cc. of a morning specimen injected intravenously into marginal ear vein on each of two successive days.
Time	Forty-eight hours.
Endpoint	"Blutpunkte" (hemorrhagic follicles) or corpora lutea formation (shown in drawings next page).
Accuracy	97-98 per cent.

ASCHHEIM-ZONDEK TEST

Animal	Immature female mouse 3-4 weeks of age and 6-8 gm. in weight.
Urine	2 to 3 cc. of an unconcentrated morning specimen is administered in nine equal doses over a three-day period.
Time	Ninety-six hours.
Endpoint	"Blutpunkte" (hemorrhagic follicles) and corpora lutea in the ovary.
Accuracy	96-98 per cent.

HOGBEN-FROG TEST (African Clawed Frog)

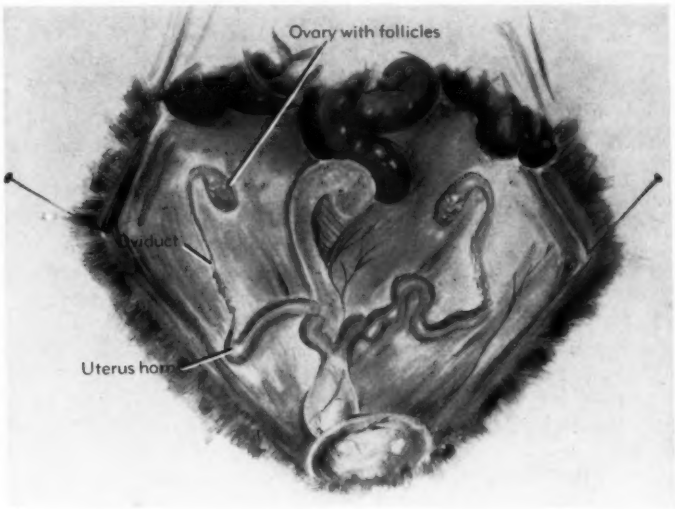
Animal	Adult isolated female frog.
Urine	80 cc. of a morning specimen are concentrated by acetone precipitation. The dried powder is taken up in 2 cc. of water. Aqueous extract is injected into the dorsal lymph space.
Time	Animals are observed from 6 to 12 hours.
Endpoint	Extrusion of eggs into water of tank.
Accuracy	98-99 per cent.

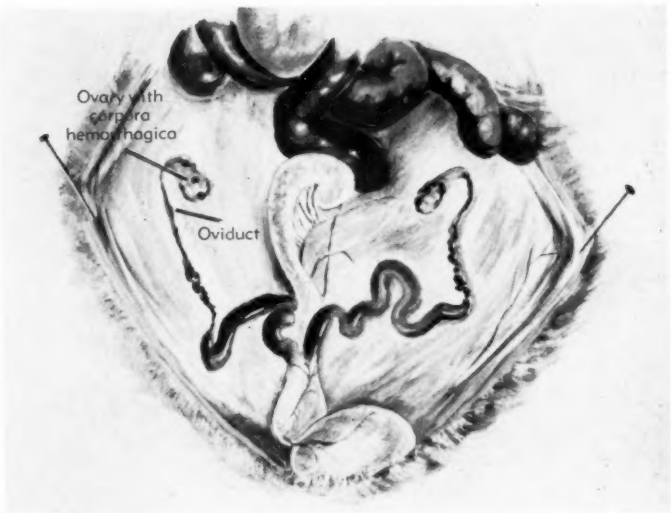
The accuracy of the two-hour test performed on urine samples from more than 1,200 patients exceeded that of the three procedures most commonly employed in the laboratory diagnosis of pregnancy.

The sketches below of the reproductive system of rabbits injected with normal and pregnancy urine, respectively, are presented to show differences in diagnostic endpoints between ovarian hyperemia employed in the two-hour test and corpora hemorrhagica obtained in isolated female rabbits injected with urine of pregnancy.

Negative

Note follicular development only.





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The Medical Bookman

PSYCHIATRY IN A TROUBLED WORLD*

DR. MENNINGER dedicates his latest book to three groups: to the many millions of G.I.'s who furnished the basic facts for this study; to colleagues in psychiatry, both in and out of the Army; and, finally, to those military officers in every Division and Section of the War Department who helped toward the accomplishment of the "mission."

If anyone cares to ask, why write a book like this so long after the shooting war has ceased, the answer is that it could not possibly have been written sooner. As it now stands, some eighteen months were required to put it into print. Another reason is that psychiatry has expanded and developed as the result of war incidents to the point where it is no longer in "the rear seat in the third balcony . . . but in the front row at the show."

The first part of the book is written for the record, to set down what happened, why it happened and the conclusions to be drawn therefrom. Part II is concerned with psychiatry in peace, and hopes to be helpful to anybody who has to get on with other people; that means with the whole world in general. It is this section which will have the greatest appeal to most readers because it drives home certain facts which have evolved from the experiences of those who went to the front, and of those who remained here. The return of the soldier to his home, family, and friends was an event of the greatest importance to all concerned—not because he got back "safe and sound" in most instances, relatively speaking, but because of the inability of both combatant and noncombatant to adjust to each other. Here we have set before us the ultimate in environment and social conduct—the loving mother who has wept and worried and who loves her son better than her own life, who rushes to the swinging gate to fall upon his shoulders only to find him strange, even indifferent to her approach.

The father too finds that "Joe is different." Under Army discipline, the soldier was made to obey. He was always under somebody's watchful eye and, therefore, he became an automaton, moving this way or that like a puppet on a string, but now that he is "out," he remembers the authority of his father's restraints. They still rise up to plague him, but he is a man now, and out from under anybody's yoke—he will "show the old man where he gets off!" Personality clashes ensue with the result that parents and son are all unhappy; for "things have changed." A boy in his teens, still in high school or early college days, swung onto the last step of the disappearing train, waving merrily good-bye—off on a "lark," so naïve is youth. But the rigid training to kill his brother in war, the narrow, uncomfortable tent (without running water and toilet facilities), the anxiety for his buddies, the death and destruction always round about—all these have converted an innocent lad into a hard-hearted, devil-may-care, unemotional animal, who has become prematurely aged beyond his calendar years.

There are also the wife and child. Pictures of his baby born while the soldier was absent have been shown to every buddy or chance acquaintance with great glee, but now the child is 3 or 4 years old, refers to "that strange man," and is not easily influenced by him. There may be here more than a mere touch of jealousy and distrust. How is the veteran going to be sure that the "brat" is not from the loins of another man? The marriage, the very few nights together, the hurried departure gave no time for a blending or moulding of the young lives, and the "love" from which their sexual act sprang, may it not have been spurious and altogether untimely?

*Psychiatry in a Troubled World. Yesterday's War and Today's Challenge. By William C. Menninger, M.D., General Secretary, The Menninger Foundation, Topeka, Kansas; Chief Consultant in Neuropsychiatry to the Surgeon General of the Army, 1943-1946. 636 pages. New York, The Macmillan Company, 1948. Price $6.00.

And, again this matter of sex—the soldier "takes his fun where he finds it," be he officer or the lowest in rank. Very often he cannot even be discreet about the act, and promiscuity is known and even bragged about in the barracks. It is impossible not to think that what is going on in camp may not also be going on at home. "The wife has feelings too." The single standard maintains in books and in refined conversation, but it is not analyzed in the mind and heart of the one who is left starving for affection and companionship. It is a combination of all these circumstances which makes of War the hideous, loathsome, insane thing that it is. Men cry and have always cried Peace! Peace!! and yet there is no peace—merely an armistice, a hidden, sullen feeling of rage against the "enemy" who happens to have been born in a different locale and of a different race and creed.

All this is true and recognizable in the story of "civilization" from its first crude beginning. Man is still closely akin to the beast with all of the primeval instincts of the savage lying close beneath his social veneer. Long before the Bible was put together to be "the guide and stay" of those who seek for a better life both here and beyond, Man was doing awful things to his Brother, which were not very different from what he is doing today. And, speaking of the Bible, that great book contains some of the worst horrors in history, some of the most revolting practices recorded in the "doings" at Sodom and Gomorrah (still revolting in this day and age, and still being practiced). The Book of Leviticus, to mention only one, is replete with these vulgarities, not to use a stronger word. All of these facts, however, do not condemn nor destroy the influence of the Bible as a great book, but we can weed out the undesirable elements just as we do when reading some of the present-day pornographic novels, many of which are "well written" and manage to carry a message of good in the long run.

But to return to Dr. Menninger's book. Chapter 28 records the "defects revealed in civilian medicine" which have been carried over into Army regimentation, and in the passing have been magnified and no doubt worsened. There is a lack of integration; a separation of civilian from military medicine to the hurt of both. Regular Army men "kicked around" the individual doctors who came from positions of great independence in thought, word and deed to "under-the-thumb" orders of the "line organization men," many of whom had had no medical training whatever. There were (as always) an enormous number of individuals who *knew how to win the war,* and insisted on making everybody else conform. When and where they had authority to impose their will, the going was pretty "rough" for the highly-educated, gentlemanly man of medicine. Specialists were not always regarded with favor by the Regulars; therefore, they were sometimes kept idle for months in the very presence of an urgent need for "more doctors" in the right places. Remedial or curative medicine is the *summum bonum* of every well-trained civilian doctor, but he often found himself doing "paper work" in screening, planning and administration.

As for psychiatrists, they were swamped by thousands of referred recruits who could have been helped to adjustment by general practitioners. In civilian life these G.P.'s manage to advise men and women of all stations in life, and help them over the hurdles which are quite often not too high for ordinary intelligence to negotiate. However, the medical schools seem not to have done their job in teaching even the elements of psychiatry; for the "physical" of no patient is entirely complete without a satisfactory survey of his physical make-up. Lack of recognition of this principle may account for the enormous talk about psychosomatic medicine so much in the medical literature these days. It is by no means a new thing. Our grandfathers had it, and perhaps even in greater measure, though crude, than we have had in so-called "modern medicine."

Chapter 35 of some 10 pages is devoted to Research in Psychiatry. There is a crying need for well-trained psychiatrists who must take up the burden of the stress and strains of postwar hysteria and environmental uncertainty. It seems that in spite of all our great strides in technology, economic studies, research in physics and chemistry, and the social sciences, we do not yet know how to live happily on this mundane sphere. Unless we *do* learn, we shall probably go on from good to worse, from a deep and appreciative consideration of the humanities to "murder as a fine art" which takes up so much valuable time and space in the movies and on the air. Dr. Menninger's book, if carefully followed, can do much to set us right.

I. W. V.

MEN OF MEDICINE

Scientist, Philosopher, Humanist

This biographical sketch of Dr. Myerson was scheduled for publication before his final illness. He was, however, already quite ill when he graciously consented to interviews with *Postgraduate Medicine's* staff writer. Shortly afterward, and before the article was completed, Dr. Myerson died in September of this year. *Postgraduate Medicine* is pleased to offer this article not only as a tribute to his outstanding career, but also as an impression of Dr. Myerson in what proved to be his last weeks of life.

IT MAY be a matter for controversy whether or not we are indeed a generation of vipers, but there can be little argument that we are a glib and garrulous generation. We can qualify, rationalize, evade as ingeniously and as futilely as any medieval theologian, but we shy away from anything that rings with forthrightness. Seldom do we trust words or concepts that have at their core, in spite of the abuse and accretions of time, an undimmed lustre. Such are the simple words *great* and *good*. Yet Doctor Abraham Myerson, both as man and as physician, truly merited them both.

Undoubtedly his humility and his skepticism would have made him smilingly reject the tribute, but a man's life speaks more forcefully than a disclaiming phrase—a truism that Dr. Myerson could not disavow, for he said of himself, "I am a skeptic in many of my *beliefs,* but not in my ways of life. For I believe that willy-nilly man will continue to live on this earth; that somehow I am bound to my fellows so that I wish them well and must work for and with them."

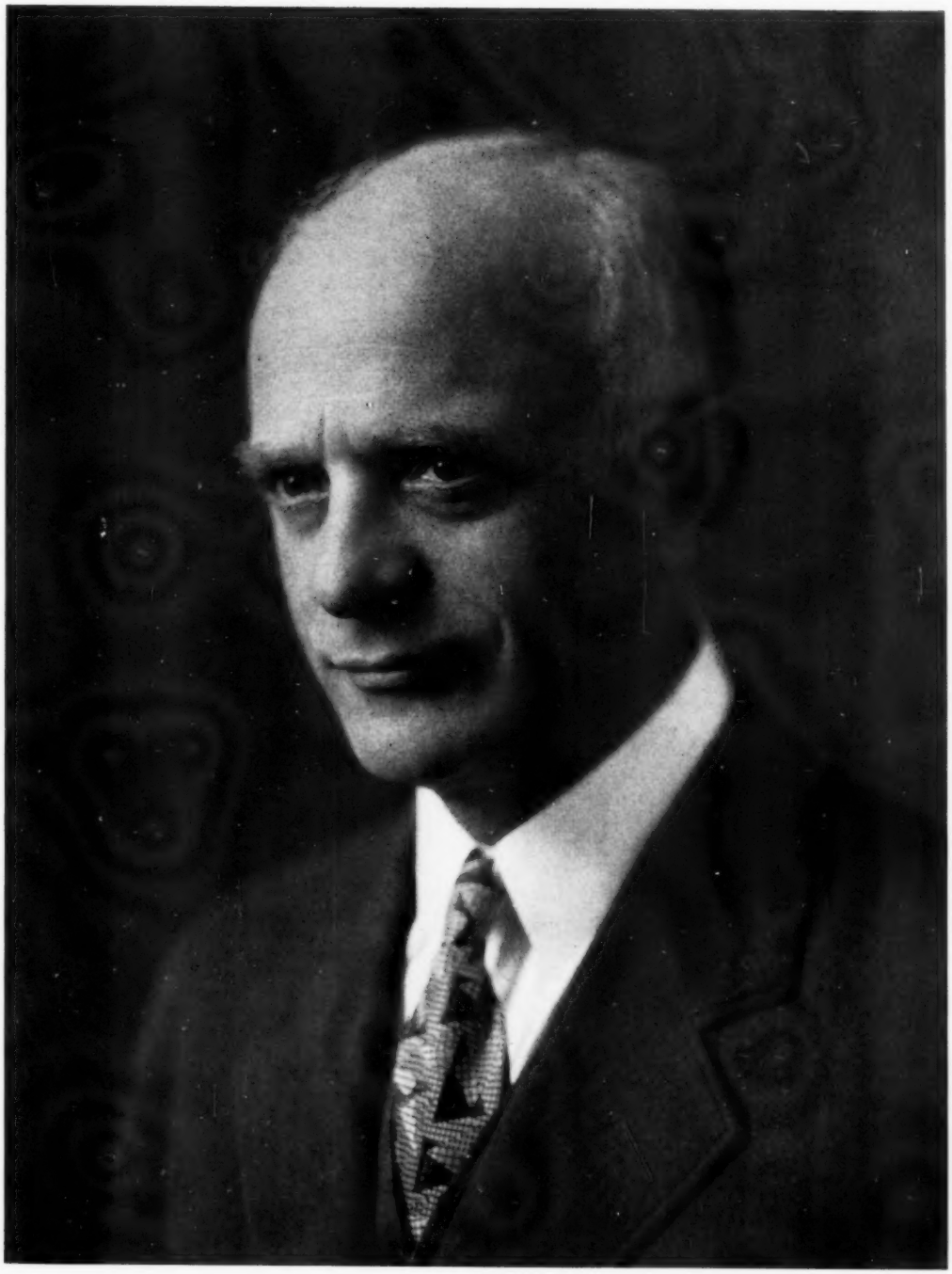
Late in the summer of 1948, those long years of strenuous work with his fellow men had etched their story in Dr. Myerson's drawn, sharpened features, in the emaciation of his always slight frame. But the deep set, searching eyes still burned with fervor as his mind pursued its lifelong quest of the answers to those imponderables: What is Man? And how is Man to achieve wisdom?

Throughout his maturity he had studied those problems, and in September of this year he died, unsatisfied with the answers he had been able to formulate. Nevertheless during the last year of his life, often bedridden and in pain, he was working on a book by means of which he wanted to share with others some of his conclusions about what constitutes the worthwhile life. Fragmentary and unfinished though it be, that volume is a source of comfort and a friendly guide toward further thought for all who may read it. He claimed little for it, referred to it lightly as a "book of digressions," but in it with deep sincerity he has distilled those convictions, founded on experience and knowledge, subtly colored by temperament, that made his life so significant.

It is not to be wondered at that a neuropsychiatrist should produce such a book, nor that all his years should have been preoccupied with the question of the nature of man. What better approach is there to that enigma than that of the trained scientist who has worked for more than two score years with that "blood-soaked sponge," the human brain? To that brain, which is man's main instrument of knowledge, come a few sensory avenues by which he can learn about the world. But let Dr. Myerson's own words speak for him.

> I have dealt with that blood-soaked sponge, man's brain, for forty years as a professional neuropsychiatrist, and I have tested its functions during life and studied it after death chemically, microscopically, and diversely. I marvel at it and respect it, but it is a very limited, although marvelous, instrument at best. Even with all the tools it has created, it lives in an insignificant fragment of the universe and is subject to all kinds of diseases and disasters.
>
> Indeed, it puzzles me why men, philosophers and some scientists talk of the MIND of Man in capitals, and as if it were too lofty to have any direct dependence upon the structure of the brain. These people seem never to have met an Idiot with no more general ideas than a puppy and, indeed, not so intelligent. Apparently they never have witnessed the decadence into imbecility of talented men via old age and cerebral arterial disease; and they never seem to understand that when alcohol changes a man from Philip sober to Philip drunk, chemistry and mind and brain have become knit as finely together as any chain of cause and effect in this world of ours.

Between the lines of this statement and permeating it throughout, is Dr. Myerson's staunch belief in the physical basis of mental disease. His approach to the nervous system and its ills was by way of chemistry, physiochemistry, genetics, and biology, rather than by way of Freud's neatly compartmentalized Id, Ego, Super Ego, etc.



ABRAHAM MYERSON

He made his opinion of the present status of psychiatry clear, and added his prediction for its future when he wrote in 1947:

> The present status of psychiatry, so far as much of its work and certainly most of its publicity is concerned, is deplorable in extreme measure. The loose kind of thinking that characterizes psychoanalysis is now dominant in the public eye, but I assure you that the solid work of psychiatric research still goes on and is not greatly influenced by the hulaballoo of psychosomatics, etc. . . .
>
> If I were asked to predict the status of psychiatry twenty-five years from now, I would state without hesitation that biochemistry, biophysics, pharmacological therapeutics will hold the center of the stage; that psychoanalysis will be present as a term, but entirely different as a system of beliefs and principles; and that the social phases of psychiatry with its genetic and social relationships will be of fundamental importance.

In all fairness to his Freudian-derived opponents, Dr. Myerson was quick to admit that he was certainly a combative person and perhaps a very prejudiced one. Certainly, they would so regard him. He was, however, not merely a theorist but a generous and open-minded person who could say in the humility that accompanies only deep wisdom that he was never estranged from his colleagues by differing beliefs, nor were his personal relationships of affection and esteem for them ever altered. "For," he mused, "in this vast and overpowering universe that surrounds us, crowds in upon us, weighs us down and inevitably molds us, how can any man have but a flicker of knowledge, an infinitesimal bit that he gratefully adds to the common store. Why should I assume that a portion of final truth has been made manifest to me alone?"

As a scientist Abraham Myerson believed profoundly in the power of heredity; as a man he demonstrated it. His father was an immigrant to the United States from a small town in Lithuania which he was forced to leave because his brother had busied himself with collecting a library for the Jewish community, and had been promptly exiled to Siberia for his effrontery. Once in this country, with a growing family to support, the elder Myerson wandered about as a peddler, then established a small and not particularly profitable business in Connecticut. Afterward he removed himself and his family to Boston where they settled down in the rough and boisterous South End.

The three boys learned to be cocky and effective fighters—they had to be, to protect themselves against the South Boston gangs. But at home the influence of the idealistic, scholarly father was great; congeniality and companionship flourished. "By the time I was fourteen, there was no longer any conflict between my father and me. We saw eye to eye and debated endlessly matters of philosophy and history. But the odd thing was that I didn't realize there was a chance for me to become a professional man, to make something of myself until through helping my brother Joseph in some business venture, I became acquainted with some poor but very able young intellectuals in the West End of Boston. Then, suddenly, ambition spurred me to become a scientist. Why not? My older brother, after selling newspapers at twenty-two, had become a doctor. And wanting to be a free agent, hang out my shingle, and sink or swim by virtue of my own ability, I chose medicine, too.

"Of course, it wasn't as simple as that. I kept running out of money. I had to leave Columbia and come back home to Boston. I worked as a streetcar conductor. Then I managed a year at Tufts Medical School. More money troubles, and another year at Columbia. Then at Tufts I found a friend, Morton Prince, the eminent neuropsychiatrist. I was good at brain anatomy and I liked it. Morton Prince encouraged me. . . . Much later I held the chair of neurology that Prince had had."

And the amalgam of Jewish scholar and Boston's intellectual atmosphere produced a man both wise and great. Just how they were combined in one individual may best be made clear by a Jewish folk tale or fable, of which Dr. Myerson said: "This was my father's favorite story."

> There once took place a conclave of scholars, and they discovered that no one had ever defined what a scholar—the word and the man—really was; so they set about making a definition. One man said this and another said that, and there was a very lively scholastic debate until an elder arose to finish the discussion.
>
> "A scholar," he said, "is like a pin which must, as you know, have a good sharp end but, likewise, a good round end. The scholar's sharp end is to think with, his good round end is to sit down to the job with. If he has only the sharp end, he becomes a dilettante flitting from job to job, accomplishing little. If he has only the good round end, he is a pedant, unimaginative, uninspired, and learning only by rote. Yes, the scholar must have a good sharp end and a good round end."

It would be hard to find a better modern instance of the type of wise man described in that story than Dr. Abraham Myerson himself.

Perhaps one reason that he felt himself so much in harmony with Boston was that like so many of the great sons of that city, he burned with a crusading fire to help people, to make them better. But never did that zeal deny the claims of the flesh, nor labor at twisting human beings into patterns they could not maintain. Instead he worked with his patients to help them live more happily within the framework of their own limitations and handicaps whether mental, nervous, or functional.

No one who ever saw Dr. Myerson with his patients, whether in the wards of Boston Psychopathic Hospital or in the quiet restfulness of his office overlooking the Charles River Basin, could fail to be impressed by his tolerance, insight, and sympathy. Because he accepted the biological facts of life, he could help others to recognize them and to cease a futile and disintegrating rebellion.

"You see," Dr. Myerson was wont to say, "we have to deal with what people *are*—not merely with what they *do,* or even what they *become.* Heredity is tremendously important—probably the most important subject in the whole field. And there's no sense in always asking whether it's more or less important than environment. We contrast the two factors for convenience in speaking but actually we shouldn't make a dichotomy where none exists. We're too inclined to talk as though the two elements are diametrically, unalterably opposed, when as a matter of fact they're just different facets of the same thing. . . . You can't classify people in a card index, or divide personality into noncommunicating compartments as did Freud. Life and living people just aren't like that."

And Dr. Myerson, even in the last grim days of his own pain and illness, was never one to forget what living people are like.

"Next to heredity," he mused, "probably my greatest interest has lain in the search for humane drugs that will affect the autonomic nervous system. Drugs to lighten the mood, lift it from black depression to normalcy. For years we've carried on research and experiments at the Boston State Hospital—we did a great deal of the early work with benzedrine sulphate there. And in my laboratory too; many a time I've been the guinea pig myself—and aside from making the patient happier, we must find a drug to give him sleep, restful sleep, not narcosis.

"All the tension and conflicts of our life today are in a conspiracy to destroy the normal mood, to banish sleep and rest. . . . Perhaps physiologically, sleep is an anemia of the brain, an autointoxication caused by fatigue. We do know that it is the way the organism remarshals its energies for activity. Like the appetite for food, it is caused by a breaking down of the tissues, and very likely this breaking down is to a large extent in the organs of special sense—vision, hearing, feeling, etc.

"Consciousness is essentially a product of the activities of the special senses. Yet for recuperation we must sleep. Without sleep, the burden of consciousness weighs too heavily upon us. Sleep helps all our conflicts; without it there is no enjoyment of life; fatigue kills everything, makes us incapable of realizing any satisfaction in life. Such a life is intolerable, but sleep keeps away anhedonia, and gives us respite from the intensity of modern life. Very early in nearly all psychological disturbances the sleep mechanism goes wrong, and then fatigue poisons life and makes it bleak and bitter. . . ."

Dr. Myerson's last illness was complicated by sleeplessness, and the fact that his system rejected sleep-inducing drugs as toxic. His suffering shadowed his eyes and vibrated in the tones of compassion in his still eager voice.

"And then there are the popular books on psychology—usually fallacious in their optimism and their certainty of knowledge, whether that certainty be expressed or merely implied on every page. You should remember that in all science, and especially in medicine, the optimists write by far the most because pessimism doesn't pay dividends or royalty checks. Thus, if you'll just learn from me 'how to relax' says one man, you'll be all right mentally, which is like saying that if you gargle your throat, you will cure sore throat, pneumonia, and cancer. Besides, in most mental illnesses, the real trouble is that the relaxing capacity is as much impaired as the integrity of a bone is destroyed by fracture. . . ."

He distrusted all authoritarianism, whether it was the garbled panacea of the popularizer or the pronouncement of the traditionalist vested with the weight of religion. Some of his most telling blasts against such gentry steamed forth from his reviews of psychological and pseudo-scientific best-sellers. Flanders Dunbar's book *Psychosomatic*

Diagnosis, which presents the theory of predisposition to accident because of conflicts in the personality, offered a particularly good target. As might be expected Dr. Myerson pointed out the fallacy of drawing deductions from the specific instance with no proper control group.

Thus the statistics in regard to accident overlook the great statistical facts about accidents. . . . Furthermore, there are alternative explanations which are not even considered in this book, and which may seem incredibly naïve to its author. First, nothing is said of the recklessness of character. . . . Another explanation is also too simple for words. Nothing is said of the relative skill, coordination and bodily manipulable characteristics of the individual. . . . To make this study of accident at all valid, tests should have been invoked and results coordinated with accident potentiality.

Psychosomatics is valid insofar as it states that mind and body are one; that what we call states of mind always become translated into bodily events; and that emotion, mood and even thought become organic. . . . It is undoubtedly true that there are changes in bowel and heart activity, to cite two functions of many, in which training, conditioning, and the life circumstances, which we call psychologic, may alter bodily structure in somewhat the same, although in lesser measure, than do food, oxygen, vitamins, chemicals and minerals of all sorts. Why not? But to select any single psychological event or psychological trend means that one has excluded all other possibilities or, more cogently, all interactions including those of heredity, physical structure, diverse and conflicting personality trends, as well as chance itself. But if you are wed to a theory, you cannot see any other factors save those congruous with your hypothesis. The paradox remains: bodily states are really greatly altered by mental events, yet much of psychosomatics is nonsense.

From such outspoken expressions of opinion it is clear that Abraham Myerson was not afraid to differ with popular views, be the question one of appraising a best-seller or bringing up a child. True, he could differ tactfully, but there was no pussy-footing around generally accepted dicta. At a child welfare meeting in Cleveland where he gave a paper he frankly announced that he did not see eye to eye with the chairman in the matter of raising children in an unadulterated atmosphere of sweetness and light. He went on to make his point clear.

Before the child develops an individual conscience he must be guided by the praise and blame, reward and punishment of his elders and those around him. . . . The child has no standard of his own, at least at first, except that on the whole it is pleasurable to get what he wants, but this becomes unpleasant if associated with the displeasure and the punishment of society as represented by his parents. The reins by which the child must be guided are praise and reward, blame and punishment, and each of these is essential. . . . But life has a habit of punishing for faults or mistakes. . . . It is surely better for the child to learn that pain follows nonconformity and unsocial conduct at the hands of those who love him than to defer that lesson and learn it from those who do not love but will be malicious in their punishment.

But in his later years the doctor recalled with a rueful smile his enthusiastic spreading of the gospel when his first son was born, his eagerness to instruct other parents.

"Why I felt practically like Moses with the Tablets of the Law! I set up *Ten Rules for Parents* and made a big noise about them. Paul turned out to be a fine youngster too. And so did the second boy, David. But somehow he reacted differently to my training. I became less sure of my ground, began to make reservations. I changed the title of my lecture to *A Few Suggestions for Parents.* Then along came my third child, a daughter. She defied every rule and suggestion I'd ever formulated, but strange to say she grew up wholesome, normal, and charming besides. You really should meet Anne—so I just subsided into the role of silent observer."

Anne and her father were always close. Very often she was his companion on the casual kind of salt water fishing trip they both loved. Dr. Myerson described how they liked to fish.

"There was never any of this elaborate equipment and detailed plan of campaign. I just wanted to catch the fish, not fight with them all afternoon! It was enough to be rocked gently in a boat and watch the sea shimmer and gleam clear to the line where it touched the sky. Sometimes we'd see a school of fish ripple the surface. And there were the gulls, darting and swooping restlessly above the water—That's how I liked to fish.

"And as I got older I expected the same relaxation from my other hobbies. I don't want my pleasures competitive or tense. There's too much unavoidable tension in the world today."

Dr. Myerson's devotion to his family was deep and unfailing. His wife, a talented artist and painter, he never ceased to woo. "But then, she's a very remarkable woman. She can cook you a meal,

or paint you a picture, or decorate your house, or entertain your friends." She is, indeed, a talented artist and painter whose gracious spirit and unerring taste are reflected in the dignified and spacious home in Brookline where the Myerson family life centered.

Anne has been studying social work at Simmons College where her father used to lecture on the psychiatric phases of social work; David, resident at Boston Psychopathic Hospital, is specializing in psychiatry; Paul, the eldest son, is a practicing psychiatrist and psychoanalyst. That last word might well have been anathema to the anti-Freudian Dr. Myerson Senior, but so broad and tolerant was the understanding between the father and son that no conflict or bitterness ever arose; the two doctors shared not only the pleasant suite of offices on Bay State Road, but in recent months the responsibility for patients besides.

"Of course, during the peak ten years or so of my practice, I saw probably 1,500 patients a year. Naturally many of them were in clinics, in state and city hospitals. And a psychoanalyst treats rather less than a dozen patients a year. Society isn't going to be healed very fast at that rate."

Its strictly limited social usefulness was but one of Dr. Abraham Myerson's criticisms of the psychoanalytic technic. More fundamental was his charge that there are no authenticated cases of the cure of serious mental illness by psychoanalysis alone. He admitted its usefulness in helping those who felt themselves bound in by their own limitations of character; such people could perhaps become better sculptors, housewives, columnists. They could, that is, if they had two or three years and several thousand dollars to devote to the adjustment of their personalities. But he was extremely skeptical of any claims that fully developed cases of paranoia, manic depressive states or severe anxiety and obsession states could be cured.

Judicious and modest in pressing his own view, Dr. Myerson ruminated on the question: "Just recently there's been one man—John Nathaniel Rosen—who claims to have cured an authentic case of schizophrenia by psychoanalysis—I won't say it's impossible."

Actually this stand on psychoanalysis was but one easily publicized strand of his fundamental disagreement with Freud and his theories. Dr. Myerson, philosopher by temperament and inclination, scientist by conviction and training, recognized Freud as a genius (a term he carefully withheld from most of Freud's followers and interpreters) and an artist, but not as a scientist. In a criticism of Freud's theory of sex, he wrote:

Like an artist, he transforms diversity into unity by suppressing those manifestations of reality which are to him irrelevant and he boldly passes beyond proof, labeling as certainty what is merely opinion.

Like a propagandist fighting fiercely against sham and insincerity in sexual matters, he cites the positive instances only, that is those facts and cases which suit his purpose. The numberless cases which might refute his theories receive scant or contemptuous mention, while his attitude toward opponents is not at all that of a man of science, but distinctly that of the leader of a cult.

Like a mystic, to Freud everything becomes symbolic of something hidden, and nothing is what it seems to be. But the symbolism is naïve since all things somewhat straight symbolize one sexual organ, and all things somewhat round symbolize another.

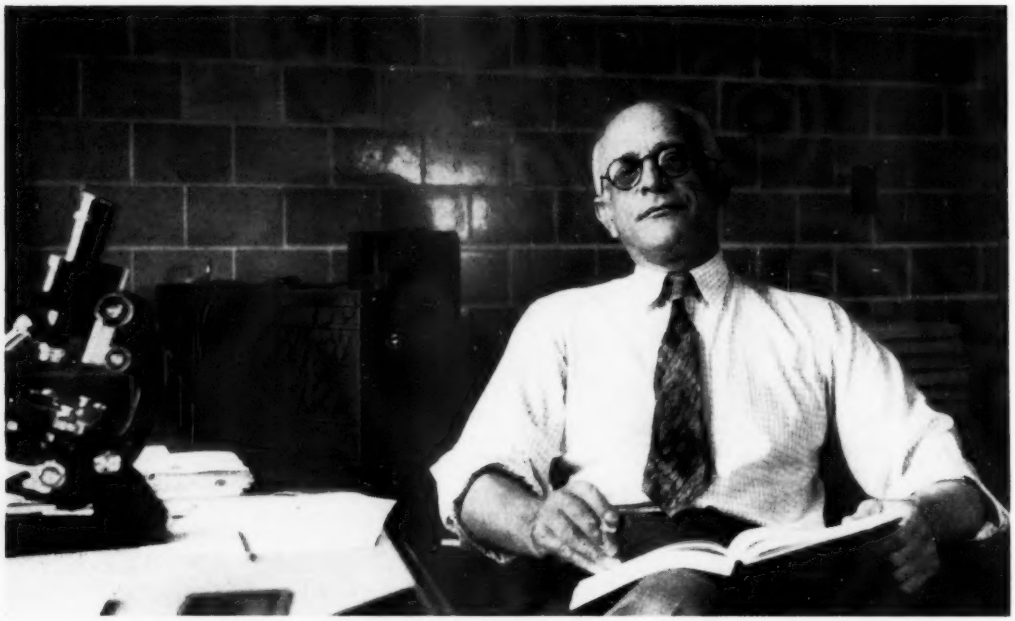
POINT by point Dr. Myerson went on to refute the Freudian theories of sexual perversion, infantile sexuality, of the Oedipus complex or incest motive, and finally the analysis of dreams by the free association method. Where there is so much danger of misinterpretation, it seems fairer to let Dr. Myerson state his position in his own words.

It is impossible to discuss the validity of the free association method by which the dream shows the Oedipus complex, the father fixation, that fantasy about the mother with all the tangled skeins of lustful love and murderous hate, transference, resistance, and the stock in trade of psychoanalysis.

Categorically one must state there is no free association method, that the psychoanalyst conditions most of the responses he gets, selecting those which suit his preconceived ideas. Moreover, you can get any complex you are after just as well by ten words selected at random from a time table, as by the episodes of the dream itself. This is not an idle statement, for I have done it. It is pertinent to remark that in the first few years of my neurological experience, I sought diligently to become a Freudian, so I am not talking as one who has always been a skeptic.

There speaks the scientist whose common sense endeared him to many and commanded the respect even of those who disagreed. Too often the cloak of plain speaking is boorishness, but Dr. Myerson is meticulous in judgment and graceful in praise.

It may seem presumptuous for anyone but a Havelock Ellis to criticize Freud since these are the two names most



DR. MYERSON IN HIS LABORATORY

associated with the study of sex in our times. Yet the technic of science is something which even the humblest may understand, and anyone whose daily work brings him the same contacts as those through which Freud himself has established his doctrines may properly criticize both the technic and conclusions of this great man.

History records many eminent men who have been as eminently wrong. The genius of Freud resides more in his influence on mankind than in his direct work. Right or wrong as his conclusions may be, or as time will judge them, he has forced us all to dig more critically, explore more candidly into human morals and human conduct. He has helped break down sham by tearing away its cloak, shame. He has made what we foolishly call obscenity a matter which we may study as objectively as in botany we study the stink weed and the rose. He has contributed to the result that all the phases of sexuality are losing their aboriginal powers to cripple and maim our grip on reality. Any man who has this as his achievement deserves the esteem and gratitude of all mankind, of those who reject his specific doctrines as much as those who accept them.

DR. Myerson was quick to recognize how great is our ignorance of the origin and progress of mental illness. "The sad fact is that we're still very ignorant of much that constitutes mental health and mental disease. . . . Our field is one in which theories reign; it is the least accessible to scientific method, largely because the mind of man offers no field for experimentation comparable, let us say, to the functions of the liver."

Dr. Myerson was ever eager to pursue the scientific method, to study facts rather than speculate on theories. At the end of his extraordinarily active and useful life, there was one accomplishment that seemed to him more important than anything else.

"It seemed theoretically correct that if one could study the blood directly before it reached the brain, and then could study it directly as it came from the brain, without admixture with venous blood from other parts of the body, something might be learned of what takes place within the brain."

It proved possible to obtain blood from the internal carotid artery without cutting down on any of the branches that lead to the face and neck, by the use of the ordinary Wassermann needle and the Luer syringe—a technic which, though it is without danger, looks formidable. Actually it is easily learned and entirely practicable.

Used together with a spinal fluid examination, the technic seems a complete method for the study

of brain metabolism. In addition the results of disease processes within the brain may be ascertained in some measure by this combination of jugular and carotid puncture.

This contribution, reported in the *Archives of Neurology and Psychiatry,* as "Technic for Obtaining Blood from the Internal Jugular Vein and the Internal Carotid Artery," provided a new method of studying at first hand the metabolism of the brain, and was developed in collaboration with two other Boston doctors.

Estimating such a skilled but purely mechanical procedure as his greatest contribution to the field shows clearly the direction from which Dr. Myerson expected progress in the successful treatment of mental ills. That others shared his beliefs was evidenced by several of the papers presented at Washington, D.C. in May 1948 at the second annual scientific meeting of the Society of Biological Psychiatry.

Another way of indicating the same road was Dr. Myerson's use of the electric shock treatment in depressive states of old age and psychoneurosis. Incidentally, though the procedure was originated by two men in Italy, then tried in England, and introduced here by Dr. Lothar B. Kalinowsky of New York, Dr. Myerson himself did a great deal of pioneer work in the treatment, teaching it to state hospital staffs throughout New England. After seeing its beneficial results in hundreds of cases in state or city institutions, and in selected private cases, he observed that it worked exactly as successfully when given as routine treatment without saying a word to the patient, as it did when used as an adjunct to carefully controlled interviews and elaborate explanatory build-ups. Such a view held no cynicism; it was logical to expect it from the author of a study: *The Treatment of Hysteric Amnesia by Purely Pharmacologic Means.*

His colleagues in the profession, whether they agreed or not, were firm in their respect and warm in their affection for Dr. Myerson. Their respect was based partly on the fact that by instinct, training, and experience he was a very fine doctor.

"He'd listen to a catalogue of aches and pains as long as your arm, and somehow go right to the heart of the matter. He didn't wear blinders because he was practicing psychotherapy. As a diagnostician he was tops. . . . And in spite of his theories, sincerely held though they were, he leaned further toward Freud in interviewing and treatment than you'd suspect from reading his published articles. He was never one to pass out a few nembutal pills to a woman who complained of sleeplessness without asking some pretty searching questions about her sex life!"

Patients, whether they came hesitantly to the dignified office on Bay State Road, or faced the slight dynamic figure in the drabness of a public institution, were quick to respond to his warmth. To him they poured out the gnawing anxieties and shamefaced fears they had long buried even from those closest to them. Dr. Myerson's personality was warm, open, forceful. He had a photographic memory and an enviable ability to pierce through the tortuous superficialities to the very seat of the trouble. He had confidence in his patients, inspired confidence in them.

Sometimes the situation demanded ingenuity as well as confidence. There was, for example, the case of the female patient who, typically in love with her psychiatrist, craftily locked herself in the bathroom of the office suite overnight so that she could seduce the doctor the next morning. All attempts to get her out were futile; her well-known name forbade forcible ejection by the Boston fire department. The doctor arrived; he smiled thoughtfully, and tapped gently on the door. Silence within while he murmured softly through the panel. Promptly the door was unlocked, the patient eagerly came out, and happily went down to the automobile waiting to take her to the Boston State Hospital.

"But Dr. Myerson, what on earth did you say to her?"

The doctor's wise smile deepened; his clear eyes twinkled. "I just asked her how she could possibly consider eloping without shopping for a trousseau first."

The adjustment of the individual to society was a problem that claimed his attention early and that never ceased to interest him. To him it was not merely a question of how this harmonious adjustment could be brought about, but whether it ought to be done at all—the whole question of the advisability of fitting man, basically an animal creature of instinctual drives and urges, into a complicated framework of forces that reach backwards to the earliest days of the race. "The dead hand rules,—yes, and the dead thought, belief, and custom continue to shape the lives and character of the living. The invention and development of

speech and writing have brought into every man's career the mental life and character of all his own ancestors and the ancestors of every other man."

This cleavage between what man is and what society expects him to be never ceased to stimulate Dr. Myerson's thinking. In the last months of his life he found himself extremely depressed by it. "A society that twice within a man's lifetime can erupt into war so violent that it comes close to destroying all that men have so laboriously built up—a society that now holds in its awkward grasp the weapon with which it can annihilate itself—how can man—or man's pitifully primitive nervous system—adapt itself to living in such vast and overreaching shadow?"

But his despondency was perhaps a symptom of his illness. Certainly his friends and colleagues like to remember the earlier years of dynamic energy and optimistic vigor. Often he would teach in the morning—he was at one time professor of neurology at Tufts College Medical School and at another clinical professor of psychiatry at Harvard—see patients in the afternoon, squeeze in a court hearing, look in at the laboratory, visit the jails, and perhaps write a chapter or two of a book in the evening. Dr. Leo Alexander, the burly and brilliant Viennese who was first his protegé, then his research assistant and associate, and who is now his successor with the resounding title, "Director of Neuro-Biologic Unit of the Division of Psychiatric Research of the Boston State Hospital"—Dr. Alexander affectionately called him *The Happy Warrior vs. Fads and Fancies in Psychiatry*. That title is a tribute to his solid common sense; his wit and sharp lucidity were equally memorable. Dr. Myerson had "experted" at murder trials in Boston for many years. Whenever he was present the court was sure of a scientific, sensible, well-documented point of view. Experience helped him; he knew all the celebrated criminals from Sacco and Vanzetti to the traitor, Best, the malodorous radio broadcaster of World War II, who by a quirk of chance was tried in Boston instead of Washington.

Once, on being presented with that ancient conundrum, "Now tell me, in your opinion, can the defendant distinguish right from wrong?" Dr. Myerson responded thoughtfully: "Ah yes, the defendant knows right and wrong precisely as the house cat knows that it is Sunday—without fully appreciating the sanctity of the day."

Something of the same nice balance of thought characterized his comments on Sacco and Vanzetti, the famous pair whom he knew intimately, visiting them in the Dedham jail almost every day for seven years while he was doing research for the state on criminals. During that time Dr. Myerson formed a shrewd opinion of the men and their respective characters, but he was too cautious to make an unequivocal statement on their guilt.

"Vanzetti, the fish peddler, had more brains—alert and interesting kind of chap. And guilty or not, they certainly didn't have a fair trial; now if in the first place they'd had a lawyer as good as William Thompson, their last defense counsel, the outcome might have been different. That's entirely apart from the fact that they were both avowed anarchists and certainly potential killers.

"You know people are always asking about 'the criminal type.' We no longer believe in determining such a matter by cranial characteristics, but nevertheless people don't realize what an infinitely delicate adjustment of factors determines character. There are all the X's of heredity plus the accidents of what happens to anyone. Sometimes I look at a criminal and think, 'There, but for the grace of God, go I!'

"There is though a certain predisposition in that some people have absolutely no altruism in their makeup; they recognize no human rights but their own. That's a constitutional psychopathic type.

"And another thing most of us overlook is that crime is a disease of youth. If the offender belongs to a wealthy family, he's protected, everything's hushed up, time and again perhaps, until he finally learns that crime doesn't pay. Then his anxious family sigh with relief and watch him 'settle down' into a stout householder and good citizen."

Boston held for him always, as it had for his father before him, a promise and a fulfillment that never failed them. It was a feeling difficult to put into words. For the doctor in his last days it was symbolized by the gracious home in Brookline surrounded by broad lawns drowsing in the hush of late summer. "In what other city could I enjoy all this peace and privacy only ten minutes from my office? And my children have been brought up here; they love it too—and people know me—." The doctor lifted his hand toward the flowers and dappled sunlight beyond the French doors, but his eyes searched for something he could not point out.

YEARS ago as a promising young neurologist working at Harvard, Myerson answered a call from St. Louis University for a bright young man in research and teaching. There he worked under the direction of Dr. William Washington Graves, and by his ability and promise so endeared himself to his chief that Dr. Graves invited the Boston youngster to practice with him. For his part Myerson was devoted to the big, rangy Kentuckian with his gusto and breezy enthusiasm. Moreover he admired him for the way he had turned defeat into challenge. Dr. Graves had been a surgeon, a fine surgeon, but he had also been a pioneer in testing the possible beneficial uses of x-ray. In the course of that research he became sterile, his face was disfigured, and his hands so badly burned that he could no longer operate. Undaunted, he began a new course of study in this country and Europe, emerged as an outstanding neurologist.

Just about the time that young Myerson was considering his chief's offer, Elmer Southard of Harvard also wrote, inviting him to "come back and work at the new Boston Psychopathic and be on my staff." Again the New England city exerted its fascination for him. "It wasn't just the place that pulled me back. There was a certain very charming and artistic young lady. I loved both the city and the woman—not that she wouldn't have gone anywhere with me—." The possibility of even a momentary misunderstanding of the relationship he so cherished roused the doctor from his mood of reverie.

"Dr. Graves volunteered some very good advice at that time. He knew me well enough to realize that my heart called me back to Boston, and he warned me not to be a masochist—told me flatly to follow my egotistic will and benefit myself. So back to Harvard it was. And a couple of years later I was made Clinical Director and pathologist at the State Hospital at Taunton, Massachusetts. I was there four years; my oldest boy, Paul, was born there." A wistful smile curved the doctor's pale lips. "Did you know his middle name is Graves?"

From the early days at Taunton his interest in heredity was unflagging, and while admitting our ignorance of how it works—we know only that deviations from normal start in the germ plasm—he never ceased to emphasize its importance. Toward the end of his career he was consulting psychiatrist at McLean Hospital at Waverley, Massachusetts. More than one visitor to the sanatorium's beautifully laid out grounds and expensively equipped buildings has been struck by the fact that it shares with Harvard University lavish benefactions from some of New England's most prominent families.

Over the years of teaching, research, practice, and lecturing Dr. Myerson's influence was strongly felt, not only in his home city and state, but throughout the country and the world. This influence was exerted through his reports and studies of work done under his direction; through the active part he took in the projects of the American Psychiatric Association; through the many positions of honor and service he filled so ably. For years he was Chairman of the Committee for Research for the American Psychiatric Association, a job that had international as well as national significance. By his energy and persuasiveness, he got the Department of Mental Health to subsidize such worthwhile projects as new methods for the state hospitals. Indeed the whole Department of Mental Health Research Committee developed under his dynamic guidance from informal get-together meetings for discussion to a very important factor in the mental health of Massachusetts and the United States. The men discussed technics, outlined definite research problems, exchanged ideas, decided on a common nomenclature for the rapidly expanding field of psychoneurological science.

During the crowded years of teaching, research, writing, and private practice, Dr. Myerson held many positions of importance. For some years he was the chief medical officer at Boston Psychopathic Hospital, and throughout his career he acted as consulting neurologist for that institution. He served in that capacity also for Washington Hospital, McLean Hospital, Boston City Hospital. At Beth Israel he was long chief of neuropsychiatry; at different times he was a professor of psychiatry at Harvard, of neurology at Tufts. He was director of research for Boston State Hospital; an active member of the Committee on Research in Mental Health, Commonwealth of Massachusetts.

In 1935 the Rockefeller Foundation began its active support of his research laboratory, a working center of great value to the whole field of mental health. Several times he attended medical congresses abroad; he was a member of the American Medical Association, the American Neurological Association, the American Psychiatric Association, the American Psychopathological Association, and

the Phi Delta Epsilon fraternity. In addition, he bore the title Diplomate, American Board of Psychiatry and Neurology.

As his strength diminished he would have been glad to shift some of the burden of responsibility, but the depleted hospital staffs and increased demands of the war years found him valiantly responding. His health suffered from the strain, and by January 1947 he began to resign from as many of his posts as possible, though he continued to see patients even in the intervals between hospitalization periods and to within a month of his own death.

And so there came to the end of his life a man who was both wise and good—an inimitable mixture of salty realistic earthiness and a probing, restless mind. All his years he had been guided by an idealism that, with warmth and humility, embraced all mankind. He had been permeated, as had his father before him, by optimism; but at the end of his journey he was filled with bitterness that man's way was so clouded, that so little had been accomplished. He had been a fighter for the truth as he saw it, yes, but the truth had not set him free. For, to be violently partisan enough to fight, to be that far emotionally off balance, means that one automatically loses his clarity of judgment. On the other hand, if one is perfectly open-minded and impartial, he is paralyzed by that objectivity and loses the impulsive force to act. He had been a liberal—yet liberals are not tough enough to survive this chaotic, disintegrating world.

So at the end, again like his father, he declared himself a pessimist. But surely that dark mood was but a reflection cast by the suffering body on the clear spirit of one who understood the human soul. Unflinchingly he had uncovered its greed, its pettiness, its self-seeking, yet never had he despised mankind nor held himself apart. He knew that all men are brothers and he served them unstintingly. Not that they erred, but that they squandered their highest potentialities filled him with sadness.

Perhaps it is more just to summarize his views by words printed some years ago in *The Caduceus,* the Tufts Medical School year book, in which he spoke seriously to the students who loved and honored him.

When one contemplates the disturbed, even chaotic world in which we at present live, it is hard to feel that there is any essential law of progress or that mankind moves forward in a direction in which there may be both development and growth of his personal character and of a better ordered, more equitable society.

There is one field of man's activity, however, in which steady, definite progress is seen and that is *science*. Year by year man is bettering his understanding of the universe in which he lives, and is able to manipulate the forces of life in a more orderly, more useful, and at the same time, more powerful manner.

In the field of neurology and psychiatry progress seems slow and practice discouraging, if we take the short view of our work, the day-by-day outlook. But if a man takes the long view, let us say one which embraces a quarter of a century and considers what has happened in this field in that period of time, he becomes optimistic. He sees mankind, as personified by the group of men working in this field, conquering its problems, passing onward to better understanding and to better manipulation of natural forces for the purposes of prevention and cure.

There speaks the neurologist, the scientist, the disciplined but aspiring mind of a doctor great in his generation. But Dr. Abraham Myerson was also a philosopher who pondered for a lifetime the immemorial question, "What is man?"

This is how he answered.

To me Man is a thickened node in the invisible web of a universe of forces which, ever repetitively and ever anew, flows in and out of him; and he is part of an Ecology which involves plants, other animals, climate, soil, all kinds of radiant forces and chemicals. He is united by the invisible strands of Heredity to every form of life that ever lived, and his fundamental drives and compulsive activities go back to the first piece of life that ever appeared on earth.

He is immersed in age-old and ever-changing social forces which compress, enhance, destroy or deform his trends. He is beset by conflict between his biology and his sociology at every step. He is the victim and the profiteer of his cunning hands and his fatal words. Within him there rages an Inner Turmoil made up of his memories and especially words, the most important of which are I, You, We, They, and the most fatal, You, when the man learns to address *himself* by his ominous term. He is abrim with chemical factories; in fact, every cell is a better chemist and physicist than all the Nobel prize laureates put together, and somehow there is a constant and shifting balance of forces which includes hormones, ferments, enzymes, memories, ideas, emotions, moods, and all of which is an unexplainable transit from conception to that catalytic dispersal, perhaps reassemblage, called Death.

Only by remembering the range of his experience, the penetration of his knowledge, and the breadth of his compassion, can we measure what humanity has lost in Dr. Abraham Myerson's death.

Lucile C. Deinard

EDITORIALS

HIGH FAT DIETS AND CORONARY DISEASE

For some time past there has been a growing conviction on the part of many cardiologists that there is a connection between fat metabolism and intimal atherosclerosis, especially of the coronary arteries. More and more evidence, experimental and clinical, has been found to support the idea that there is a disturbance, either on the part of the body as a whole or locally within the walls of the arteries, in the metabolism of fats, especially cholesterol.

Some of the points in favor of this idea are the following: the fact that the early intimal atheroma consists almost entirely of cholesterol; the low incidence of coronary disease among certain races having diets low in fats; the increased incidence of coronary disease in persons with high blood cholesterols, whether familial or due to disease such as myxedema; the ability to produce atheromatosis in certain experimental animals by the forced feeding of cholesterol. According to Moreton, the repeated ingestion of meals high in fat may produce atheroma even though only a small percentage of the fat is in the form of cholesterol.

There are still many problems to be solved before the exact role of fat in the diet can be evaluated as far as coronary disease is concerned. It is not yet known definitely whether diets high in fat can initiate coronary atheromatosis in human beings. However, there is now some evidence that high fat diets may be harmful to those persons who already have coronary artery disease.

Although the considerable incidence of peptic ulcer in patients with coronary disease is probably coincidental, an empirical survey seems to indicate that the usual ulcer diet, rich in fats and cholesterol, tends to have an adverse effect on the course of arterial disease.

In the first group of 10 patients whose histories were reviewed, 9 were found to have peptic ulcer. All of these patients died, but their histories exhibited remarkable similarities. All the patients were male, ranging in age between 38 and 55 years, and all were known to have coronary disease. In each case, the patient died within seven months after a high fat diet was instituted. In the one case not involving a stomach ulcer, the patient was placed on a high fat diet following a severe respiratory infection.

The second group whose records were examined consisted of 17 patients who were known to have coronary disease and who developed symptoms of peptic ulcer one or more years after the diagnosis of cardiac illness was definitely established. All of these patients are living, but after they had been put on the usual Sippy regimen with its high fat content, 12 of the 17 suffered aggravated symptoms of angina pectoris within a period of one to three months after starting the ulcer diet.

It should be noted that so far we are unable to state that a diet high in fat will initiate atheroma in otherwise normal individuals. It seems highly likely, however, that the ingestion of excessive amounts of lipids may cause deterioration of an arterial wall already the site of atheroma. This is analogous with the recent finding by Paterson that in certain ani-

mals the forced feeding of cholesterol resulted in an increased incidence of arterial disease but no further damage in those animals already having intimal changes.

Although the total number of cases studied thus far is too small to justify any positive conclusions, the results were striking enough to warrant further investigation of the relationship between cholesterol metabolism and atherosclerosis and to use caution in recommending a high fat diet for patients suffering from coronary cardiac disease or persons, such as those suffering from diabetes, hypertension and myxedema, in whom there are potentialities for coronary disease. In such persons, it would seem wisest to use ulcer regimens in which frequent feedings of foods that are low in fat are emphasized.

M. P.

BACITRACIN

THE superiority of bacitracin in an ointment base over the sulfonamides, penicillin, and furacin in the local treatment of primary and secondary pyogenic infections of the skin has been reported by Drs. J. Lowry Miller and Meyer H. Slatkin and Balbina A. Johnson, A.B. The advantage found in using bacitracin was that it resulted in a lower rate of sensitization of the patient while remaining as effective as the other chemotherapeutic agents with which it was compared. Its use has been somewhat restricted by the necessity for refrigeration of the ointment to maintain its potency. Five hundred units of bacitracin per gram of ointment base was the strength employed by the New York investigators.

Comparisons of the degree of sensitivity experienced by patients treated with the different medications showed that in early use, penicillin sensitized 6 per cent of cases, the sulfonamides 5 per cent, and furacin 5 per cent, while only 0.5 per cent of patients experienced any sensitization from bacitracin.

In their report, Miller, Slatkin, and Johnson stated, "We have used bacitracin for as long as three hundred days in the same patient without producing reaction. In infectious eczematoid dermatitis and folliculitis of the beard a contact dermatitis-like reaction from medication is prone to occur. We have encountered no such reaction with bacitracin. In fact, we used bacitracin ointment successfully on a patient with folliculitis of the beard who at the time was suffering with a dermatitis from sulfathiazole ointment."

A considerable amount of experimental work was conducted in an attempt to find an ointment base in which bacitracin remained relatively stable at room temperature, which released the bacitracin when in contact with the tissues, and which was acceptable to the patient. Oil in water and greaseless Carbowax bases were found to fulfill these conditions best. Clinical trials were made of the ointments which appeared to be effective on the basis of laboratory tests. Formulas were given for two ointment bases, in which bacitracin remained therapeutically effective for at least two weeks without refrigeration.

A. E.

"IMPROVING" AGENTS FOR THE TREATMENT OF FLOURS

THE CAUSE of so-called canine hysteria, running fits, or fright fits in dogs has been known since 1946. At that time Mellanby[1] demonstrated that the responsible factor was "agene" which is defined as 1 per cent nitrogen trichloride in air saturated with water vapor. The purpose of this substance added to flour is to alter the baking characteristics.

The first observable symptoms produced in dogs placed on a diet of agene-treated flour consist of general lethargy and muscular incoordination. With advanced stages of poisoning, the animal behaves in a panic-stricken manner, throwing itself against the side of the cage, howling piteously, and clawing the air. If the hysterical animal is let out of confinement, it will run wildly about, bumping into objects which get in the way. In the severest form of poisoning epileptiform, or tonic-clonic,

convulsions may occur which can be so severe and continuous that death ensues.

Other substances such as chlorine, nitrogen, and benzoyl peroxide treated flours do not harm dogs. Apparently the toxic principle of agene-treated flour is associated with a protein fraction, and according to a recent study by Radomski[2] and his colleagues the most likely toxic principle seems to be a di-, tri-, or polypeptid absorbed intact into the blood stream.

The production of fits by a diet of agene-treated flour is not peculiar to dogs; Radomski and his colleagues demonstrated the same order of sensitivity in rabbits and to a lesser degree in cats. Rats and Rhesus monkeys, however, are apparently resistant to the manifest symptoms. The question of possible toxicity to human beings is still unsettled, but it scarcely seems desirable to include such a possible toxic agent in flour from which most daily bread is prepared.

1. Mellanby, E.: Diet and canine hysteria. Brit. M. J. 14:885 (December) 1946.
2. Radomski, J. L., et al.: The toxicity of flours treated with various "improving" agents. J. Nutrition 36:15-25 (July 10) 1948.

E. P. J.

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Medicine Makes the News

LASKER AWARDS PRESENTED FOR MEDICAL LEADERSHIP

IN RECOGNITION of their outstanding contributions in the fields of medical and scientific research, medical hygiene and public health administration, eight leaders in these fields recently received the 1948 Lasker Awards of the American Public Health Association.

Recipients of the awards for outstanding scientific research and study are: Dr. Selman A. Waksman of Rutgers University and the New Jersey Agricultural Experiment Station, and Dr. Rene J. Dubos of the Rockefeller Institute for Medical Research, New York City, jointly, for "studies of the antibiotic properties of soil micro-organisms," and to Dr. Waksman also for the discovery of streptomycin.

Dr. Vincent du Vigneaud, head of the Department of Biochemistry, Cornell University Medical College, New York, "for studies of transmethylation and contributions to the chemistry of biotin and penicillin."

For outstanding administrative achievement the Lasker Awards were given to: Dr. Martha M. Eliot, Associate Chief, U.S. Children's Bureau, Washington, D.C. "for organization and operation of the Emergency Maternity and Infant Care Program."

Dr. R. E. Dyer, Director, National Institute of Health, Bethesda, Maryland, "for organization and administration of the Research Grants Division of the National Institutes of Health of the U.S. Public Health Service."

Cited for "outstanding accomplishments in the education of the physician in the psychological aspects of the practice of medicine," was Dr. C. Anderson Aldrich, Director of the Rochester (Minnesota) Child Health Institute of the Mayo Clinic.

The Lasker Group Award this year honored: The Department of Medicine and Surgery, Veterans Administration, and especially Dr. Paul R. Hawley, its former Chief Medical Director, and Dr. Paul B. Magnuson, Chief Medical Director, "in recognition of the efficient program developed by them to provide medical care for the millions of veterans who helped in the defense of America during World War II."

WAKSMAN AND DUBOS

SELMAN A. WAKSMAN

"Their (Waksman and Dubos) studies on the relation of the association and antagonism of mixed microbes of the soil have demonstrated the antibiotic properties of soil bacteria to be of inestimable value to man's improved health and well-being," says the awards committee. Dr. Dubos' theory was that "in nature, all organic matter is decomposed through the agency of micro-organisms." He experimented with the causative agent of boils, the staphylococcus, and found another agent in the soil which attacked them. Seeking to find what there was in this bacillus that killed staphylococcus, he found the answer in a fraction of the bacillus called tyrothricin. From this, he separated gramicidin, the first antibiotic used clinically in recent years. This was done and reported on in 1939.

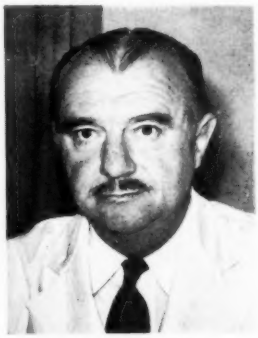
RENE J. DUBOS

After two years at Harvard Dr. Dubos returned in 1944 to the Rockefeller Institute where he is engaged in tuberculosis research. He hopes to identify that component of the TB bacillus responsible for creating the disease in man, and also the factor which causes a virulent bacillus to become nonvirulent.

The discovery of gramicidin enlivened interest in the subject of antibiotics generally. Penicillin followed,

and four years ago, Dr. Waksman and his students discovered streptomycin. The basic research that led to its development began in 1915 when Dr. Waksman graduated from the then Rutgers College and began digging holes in the New Brunswick, New Jersey, campus to isolate the soil microbes. Study of the relationships of microbes to soil conditions, decomposition, etc., laid the foundation for the discovery of several antibiotics including streptomycin.

DU VIGNEAUD



VINCENT DU VIGNEAUD

Dr. du Vigneaud is honored for "advancing the frontiers of our knowledge of fundamental living processes" in the science of biological chemistry. Out of an early interest in sulfur compounds, their structure and metabolism, Dr. du Vigneaud developed a new concept regarding the essential role of methyl groups and amino acids in the body. These discoveries are invaluable to medical understanding of the functions of the liver and kidneys. "They have given many new windows," his citation points out, "through which the chemist, the biologist, and the physician can visualize changes that characterize all living cells."

Dr. du Vigneaud is rewarded also for his accomplishments, in collaboration with others, in the studies of vitamin H, or biotin, a member of the vitamin B complex. He helped prove the identity of biotin as more than a yeast growth factor, helped isolate it, and worked out its chemical structure, all since confirmed.

ELIOT

Against deaths associated with childbirth in the United States, the nation's third highest death group, Dr. Eliot for more than twenty-five years, has fought as an advocate of improved standards of care for mothers and children. The Emergency Maternity and Infant Care Program during World War II, organized and operated by Dr. Eliot, provided medical, nursing, and hospital care for the wives and babies of men in the four lowest pay grades of the armed forces, payment for services being made by state health departments from Congressional appropriations.

MARTHA M. ELIOT

DYER

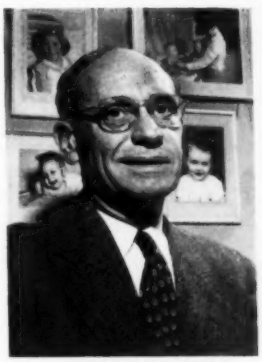
R. E. DYER

The Lasker Award to Dr. Dyer honors him in several capacities: for his scientific accomplishments in the field of microbiological research, for the administration of the National Institutes of Health during the war and postwar years, and more recently, of the Research Grants Division of the Institutes Health Service. Dr. Dyer is particularly cited for expert and impartial administration of the postwar research grants program of the Institutes which has allocated the greatest grants for medical research in world history. This program awards research fellowships, makes grants-in-aid to teaching institutions and research centers throughout the United States.

Aside from his administrative duties, which cover a period of twenty-seven years, Dr. Dyer has done considerable research on scarlet fever, typhus, Rocky Mountain spotted fever and other rickettsial diseases. In this field, he discovered that nine mile fever and "Q" fever were the same, this being one of the first steps in research on this disease. His findings that "murine typhus" could be transmitted by rat fleas to human beings, pointed the way to control of this potentially serious world public health problem.

ALDRICH

In 1944 Dr. Aldrich organized the Rochester Child Health Institute as a preventive psychiatric and well-child service for the children of the community, the first project of its kind ever undertaken. "Under his leadership, pediatricians, psychiatrists, psychologists, nursery school educators and nutritionists cooperated with city health officers, public health nurses, and the



C. ANDERSON ALDRICH

schools to discover and provide the best preventive care, physically and emotionally, for all the children of the city," according to the Lasker Award citation. The Institute was set up as a long-term project to see what could be done in a typical American community by pooling community resources for children, and developing ways of community education in physical and mental health for eventual branching out on a national scale.

HAWLEY AND MAGNUSON

PAUL R. HAWLEY

Under Dr. Hawley and Dr. Magnuson, representatives of the Lasker Group Award recipient, the Veterans Administration has six times as many full-time civilian doctors (3,659), more than fifteen times as many full-time civilian dentists (953), more than twice as many nurses (11,147), and almost seven times as many social workers (1,000) as it had in 1945. No less than the best medical care in the world for its veterans has become a reality of the Veterans Administration Medical Division by this program.

PAUL B. MAGNUSON

Dr. Hawley headed the Department since August 15, 1945, as Acting Chief Medical Director on detached service from the Army. He retired in June 1946, from the Army with the rank of Major General to become Chief Medical Director for the VA. His successor, Dr. Magnuson was formerly professor of surgery and department chairman at Northwestern University Medical School. In 1945, under Dr. Hawley, he perfected the residency training program of sixty-one of the country's top medical schools, and is responsible for other beneficial VA medical reorganizations. He played a major role in the affiliating for the first time of more than half of the Veterans Administration's till then isolated hospitals with Class A medical schools.

LASKER FOUNDATION

MR. AND MRS. LASKER

The Albert and Mary Lasker Foundation, donor of the awards, was established in 1943 by Mr. and Mrs. Lasker, to acknowledge work done in combatting the most important causes of death and to stimulate public interest and support for this work, so that it may culminate in better health and increased life expectancy for everyone.

The Awards consist of $1,000 each, and a gold replica of the statue, "The Winged Victory of Samothrace"—to symbolize victory over death and disease—for individual winners, and a silver statuette for Group Award winners. A leather-bound citation accompanies each Award.

The foundation presents awards in the field of planned parenthood in addition to those in medical research and public health administration.

New Drugs and Instruments

Information published in this department has been supplied by the manufacturers of the products described.

ADANON HYDROCHLORIDE

PURPOSE: Synthetic analgesic and antitussive drug.

COMPOSITION: Adanon Hydrochloride is 6-dimethylamino-4, 4-diphenyl-3-heptanone hydrochloride. It is a white crystalline compound, soluble in water and alcohol, but insoluble in ether. It melts at 230-235° C., and has a bitter taste.

DESCRIPTION: In analgesic doses the action of Adanon Hydrochloride is similar in some respects to morphine. In other respects it differs from morphine, producing less stimulation and showing no tendency to the development of tolerance.

INDICATIONS FOR USE: Relief of pain from various causes, particularly intractable pain of malignancy, renal colic, and fractures. Also effective in suppressing cough, especially in pulmonary tuberculosis, if its use is guarded due to possible addiction properties. It has been ineffective in pain due to gangrene.

CAUTIONS: It is a potentially addictive drug and the same precautions must be exercised as those which surround the use of morphine.

DOSAGE AND ADMINISTRATION: May be administered orally or intramuscularly. Range of dosage by any route is from 2.5 to 10 mg. The average adult dose is 5 mg., usually sufficient for control of pain; subsequent doses may be increased or decreased as necessary.

HOW SUPPLIED: *For oral use:* Tablets of 2.5 mg., 5 mg., 7.5 mg., and 10 mg., bottles of 100 and 500. Elixir—each 5 cc. (1 teaspoonful) containing 5 mg.—bottles of 473 cc. (16 fl. oz.). *For injection:* Ampules of 2 cc., each 1 cc. containing 5 mg., boxes of 10, 25, and 100. Vials of 20 cc., each 1 cc. containing 10 mg.

PRODUCER: Winthrop Chemical Company, Inc., New York 13, N.Y.

TRIAZOLINE

PURPOSE: Compound sulfadiazine, sulfamerazine, and sulfathiazole.

COMPOSITION: Each Triazoline Tablet (grooved) contains 0.167 gm. of sulfadiazine, sulfamerazine, and sulfathiazole.

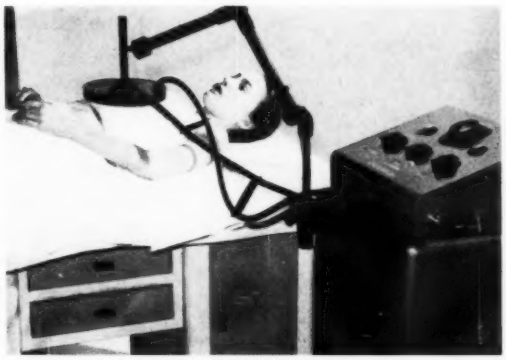
INDICATIONS FOR USE: In the treatment of any case of acute bacterial infection in which the component drugs are indicated, with the exception of meningitis.

CAUTIONS: Contraindicated in infections caused by organisms not susceptible to any of the component drugs.

DOSAGE AND ADMINISTRATION: Total dosage to be administered is the same as that which would be usual for any one of the three drugs. In adults an adequate blood level may be achieved by an initial dose of 4 to 6 gm., followed by maintenance doses of 1 gm. every four hours.

HOW SUPPLIED: Bottles of 100 and 1,000, for oral administration.

PRODUCER: Abbott Laboratories, North Chicago, Ill.



ALOETHERM

Crystal Controlled Short Wave Diathermy

DESCRIPTION: The Aloetherm operates on a frequency of 13.56 megacycles (22.12 meters), with a frequency deviation of less than 0.05 per cent. Harmonic radiation is suppressed below the 25 microvolt level required by the Federal Communications Commission.

The control panel is provided with one line switch and two control knobs, one for presetting the resonance and one for control of energy from the lowest level to the maximum output of the machine. Warm-up of the tubes is fully automatic.

The cabinet is of welded steel construction; the finish is baked, chip-proof enamel. All parts are chromium- or cadmium-plated to prevent rust. Furnished with the Aloetherm is an extra-length genuine electromagnetic Inductance Cable.

PRODUCER: A. S. Aloe Company, St. Louis, Mo.

STERI-VIALS BENADRYL HYDROCHLORIDE

PURPOSE: For conditions in which the use of Benadryl is indicated.

DESCRIPTION: A sterile solution containing in each cc., 10 mg. of Benadryl Hydrochloride (diphenhydramine hydrochloride, P. D. & Co.).

INDICATIONS FOR USE: Indicated for acute asthma, serum reactions, bee stings, irradiation sickness, and drug reactions, particularly when the condition is urgent and immediate action of the drug is desired.

HOW SUPPLIED: In 10 cc. Steri-Vials (rubber-diaphragm-capped vials), one to a package. Benadryl also is available in 50 mg. Kapseals, bottles of 100 and 1,000; 25 mg. capsules, bottles of 100 and 1,000; and as an elixir containing 10 mg. per teaspoonful, in pint and gallon bottles.

PRODUCER: Parke, Davis & Company, Detroit 32, Mich.

KIDDE DRY ICE APPARATUS

The unit is provided with various diameter plastic applicators and solid carbon dioxide snow is formed directly into these applicators ready for immediate use and confined by the active applicator tip to the lesion itself. The low temperature of the carbon dioxide snow provides its own anesthetic.

PRODUCER: Kidde Manufacturing Co., Inc., Bloomfield, N. J.

TRI-IMMUNOL

PURPOSE: For immunizing infants and children against diphtheria, tetanus, and whooping cough with a minimum number of injections.

COMPOSITION: Tri-Immunol Trivalent Immunizing Agent is a combination of Purogenated Diphtheria Toxoid, alum precipitated, Purogenated Tetanus Toxoid, alum precipitated and Phase 1 Pertussis Vaccine.

DESCRIPTION: May be used for the "booster" injection recommended for children about to enter school. Reactions are minimal and when they do occur, they are only of local nature.

DOSAGE AND ADMINISTRATION: Should be administered in three deep intramuscular injections of 0.5 cc. each for infants and older children, with an interval of four to six weeks between injections. Immunization may be started as early as 6 months of age. The "booster" injection consists of one injection of 0.5 cc.

PRODUCER: Lederle Laboratories Division, American Cyanamid Co., Pearl River, N.Y.

PETECHIOMETER

PURPOSE: For the clinical determination of increased capillary fragility.

DESCRIPTION: The device works on the principle of negative pressure applied over the biceps. After the application of suction for a period of sixty seconds, a petechial count can be made through a magnifying lens incorporated in the head of the instrument.

PRODUCER: Rexall Drug Company, Los Angeles, Calif.

NORODIN HYDROCHLORIDE

PURPOSE: A sympathomimetic drug with psychomotor properties similar to those of amphetamine.

COMPOSITION: Norodin Hydrochloride is the Endo brand of *d*-desoxyephedrine hydrochloride (*d*-N-methyl amphetamine hydrochloride).

DESCRIPTION: Norodin acts predominantly as a central nervous system stimulant. Six milligrams produces a circulatory effect equivalent to that produced by 10 to 15 mg. of amphetamine sulfate.

INDICATIONS FOR USE: In narcolepsy, postencephalitic parkinsonism, the facilitation of roentgenographic studies of the gastrointestinal tract, and the control of appetite in obesity.

CAUTIONS: Norodin is contraindicated in hypertension, advanced arteriosclerosis, coronary artery disease, and hyperthyroidism; it should not be used in hyperexcitable or neurotic patients, or in individuals who have an idiosyncrasy to ephedrine or its analogues.

DOSAGE AND ADMINISTRATION: Since dosage varies with the individual and the condition treated, it is advisable to begin with 2.5 mg. and increase by increments of 2.5 mg. until the optimal individual dose is reached. The following total daily doses, usually given in two divided doses before noon, may be considered average:

Depressive states	2.5 mg. to 10 mg. daily
Postencephalitic parkinsonism	10 mg. to 25 mg. daily
Narcolepsy	7.5 mg. to 15 mg. daily, divided in three equal doses
Adjunctive alcoholism treatment	5 mg. to 10 mg. daily
Appetite inhibitor in obesity	2.5 mg. to 7.5 mg. three times daily, thirty to sixty minutes before meals

HOW SUPPLIED: On prescription only, in 2.5 mg. tablets and 5 mg. (pink scored) tablets in bottles of 100, 500, and 1,000. It is also supplied in powder for dispensing.

PRODUCER: Endo Products, Inc., Richmond Hill 18, N.Y.

CRESATIN OINTMENT

PURPOSE: Fungicide for treatment of mycotic skin infections.

COMPOSITION: Contains 80 per cent metacresylacetate in an especially prepared ethyl cellulose base.

INDICATIONS FOR USE: In the treatment of common mycotic skin infections such as tinea circinata, tinea versicolor, tinia cruris, erythrasma and dermatophytosis or ringworm of the feet.

HOW SUPPLIED: In ¼ ounce collapsible tubes.

PRODUCER: Sharp & Dohme, Inc., Philadelphia 1, Pa.

AMPINS

PURPOSE: For administering emergency drugs, such as morphine, epinephrine, nikethamide, etc.

DESCRIPTION: The material to be injected is sealed into the special drawn ampule under pressure, using an inert gas. When the Ampin is held in a pencil position (bottom up), and the tip of the ampule broken, the inert gas forces the medication through the needle into the tissue. Just enough pressure is used to accomplish this result. The entire unit is sterilized after assembly.

HOW SUPPLIED: In packages of 5 Ampins.

PRODUCER: Strong Cobb & Co., Inc., Cleveland 4, O.

STERILE AQUEOUS SUSPENSION ESTROGENIC SUBSTANCES, R & C

PURPOSE: Estrogenic therapy.

COMPOSITION: The preparation contains biologically standardized natural estrogens (90 per cent or more estrone).

INDICATIONS FOR USE: For intramuscular injection where prolonged estrogenic activity is desired, as in the treatment of the menopausal syndrome.

CAUTIONS: Contraindicated in the treatment of women who have a familial or personal history of mammary or genital malignancy.

DOSAGE AND ADMINISTRATION: Usually 1 cc. (20,000 I.U.) injected intramuscularly once or twice weekly as required. Before withdrawal of the suspension, the vial should be shaken gently, or the contents agitated with a rolling motion to insure even dispersion.

HOW SUPPLIED: Multidose 5 cc. and 10 cc. vials, assayed biologically so that each cc. when uniformly suspended contains the equivalent of 20,000 I.U. standard estrone.

PRODUCER: Reed & Carnrick, Jersey City, N. J.

GLYTHEONATE

COMPOSITION: Each tablet contains Theophylline sodium glycinate 5 gr. (324 mg.), equivalent to theophylline U.S.P. 2½ gr. (162 mg.); phenobarbital ¼ gr. (16.2 mg.); racephedrine hydrochloride ⅜ gr. (24.3 mg.).

INDICATIONS FOR USE: In respiratory involvements such as asthma and hay fever, either allergic or secondary to bronchitis or emphysema.

CAUTIONS: Due to the racephedrine content, use cautiously in hypertension and cardiorenal disease, diabetes, and hyperthyroidism.

DOSAGE: Clinical data suggest an average therapeutic dose of 1 to 2 tablets three times a day.

HOW SUPPLIED: Bottles of 100 tablets.

PRODUCER: The E. L. Patch Company, Boston, Mass.

ELAMINE LYOPHILIZED

PURPOSE: Parenteral amino acid therapy.

COMPOSITION: Free amino acids (96 per cent), of which more than 50 per cent are amino acids essential for nitrogen balance in man.

DESCRIPTION: A modified acid hydrolysate of casein, to which tryptophane is added and from which glutamic and aspartic acids have been partially removed. The *p*H ranges from 6.5 to 7.0. The total nitrogen is 13.4 per cent, of which 75 per cent is represented by a-amino nitrogen. The product is sterile, nonpyrogenic, and nonantigenic.

DOSAGE AND ADMINISTRATION: 60 gm. of Elamine Lyophilized will keep the average surgical patient in nitrogen equilibrium. In severe cases of protein deficiency, from 2 to 4 times this dose may be given without creating untoward effects. The product may be restored to solution with any U.S.P. parenteral diluent—water for injection, dextrose solution, normal saline. It is given as a 10 per cent solution at a rate of 50 to 100 drops per minute.

HOW SUPPLIED: 12x60 gm. per bottle in a shipping carton or 6x60 gm. per bottle plus 6 bottles of diluent and 6 infusion kits.

PRODUCER: Interchemical Corporation, Biochemical Division, Union, N. J.

PRESIDON

PURPOSE: Sedative-hypnotic.

COMPOSITION: Presidon is a pyridine derivative, and is not a barbiturate.

INDICATIONS FOR USE: Especially useful for broken sleep, premature awakening, as a daytime sedative and for ambulatory cases of insomnia. With therapeutic doses, there is little likelihood of "hangover," side reactions, or cumulative effects.

HOW SUPPLIED: In scored 0.2 gm. tablets, bottles of 20 and 100.

PRODUCER: Hoffman-La Roche, Inc., Nutley, N. J.

BENZEBAR

PURPOSE: A combination of benzedrine sulfate and phenobarbital for treatment of depressive states.

COMPOSITION: Each tablet contains: Benzedrine sulfate (racemic amphetamine sulfate, S.K.F.), 5 mg. (1/13 gr.); and phenobarbital, ¼ gr. (16 mg.).

INDICATIONS FOR USE: In depressive states, particularly those following: onset of menopause, acute infectious diseases, surgical operations, childbirth; or associated with: chronic organic disease, persistent pain, old age, grief over misfortune or bereavement. These states are often accompanied by debilitating anxiety, apprehension, agitation, or nervousness.

CAUTIONS: Should not be used in patients hypersensitive to ephedrine-like compounds, or in cases of coronary disease or other cardiac conditions in which vasoconstrictors are contraindicated. Should be used with caution in the presence of hypertension.

DOSAGE AND ADMINISTRATION: Suggested dosage is 2 to 4 tablets daily, one-half the dosage on arising, and the other half before noon.

HOW SUPPLIED: In bottles of 25 tablets.

PRODUCER: Smith, Kline & French Laboratories, Philadelphia, Pa.

DIENESTROL SUSPENSION

PURPOSE: For treatment of menopausal symptoms, prevention of breast engorgement and suppression of lactation.

DESCRIPTION: Unlike oil solutions, the aqueous medium is readily absorbed without foreign body reactions or local irritation.

COMPOSITION: A sterile solution containing microcrystals of Dienestrol, ranging from 3 to 5 microns in diameter, in normal saline, with 0.5 per cent chlorobutanol as bacteriostatic agent.

DOSAGE AND ADMINISTRATION: For intramuscular injection. A 1½ inch, 22 gauge needle is recommended. When vial is thoroughly shaken, each cc. contains 5 mg. of Dienestrol. Menopausal symptoms: 2.5 to 5 mg. (½ to 1 cc.), injected in the deltoid or gluteal muscle, once or twice weekly. For inhibition of breast engorgement or suppression of lactation: 5 to 10 mg. (1 to 2 cc.), administered intramuscularly for three to four days.

HOW SUPPLIED: In 10 cc. multiple dose, rubber-stoppered vials, each cc. contains 5 mg. Dienestrol.

PRODUCER: White Laboratories, Inc., Newark, N. J.

POSTGRADUATE COURSES

Complete information may be obtained by writing directly to the Institutions offering the courses.

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

ALLERGY

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
New York Postgraduate Medical School, New York	#302—Allergy	Part time, Fridays, Jan. 7-Feb. 25, 1949	$ 25.00

ANESTHESIOLOGY

Cook County Graduate School of Medicine, Chicago	Personal Course in Ether and Nitrous Oxide (Inhalation) Anesthesia	1 month, full time, every month	125.00
	Personal Course in Ether, Nitrous Oxide, Ethylene, and Cyclopropane (Inhalation) Anesthesia	1 month, full time, every month	200.00
	Personal Course in Spinal and Intravenous Anesthesia	2 weeks, full time, every two weeks	150.00
	Two Week Personal Course in Endotracheal Anesthesia	2 weeks, full time, every two weeks	150.00
New York Postgraduate Medical School, New York	#942—Anesthesia (Specialists)	2 weeks, full time, third week	125.00 65.00 (Specialists)

ARTHRITIS

New York Postgraduate Medical School, New York	#301—Arthritis and Allied Rheumatic Disorders	Tuesdays, part time, Jan. 4-Feb. 15, 1949	45.00

CARDIOVASCULAR DISEASES

New York Medical College, Flower and Fifth Avenue Hospitals, New York	Cardiology	20 successive Wednesdays, may begin any Wednesday except during July and Aug.	150.00
New York Postgraduate Medical School, New York	#303—Clinical Cardiology	Jan. 3-Feb. 21, 1949, part time	45.00
Graduate School of Medicine, University of Pennsylvania, Philadelphia and Philadelphia General Hospital	Cardiology	8 Thursdays, Jan., 1949	80.00

CHEST DISEASES

New York Postgraduate Medical School, New York	#308—Acute and Chronic Diseases of the Chest	Thursdays, part time, Jan. 6-Feb. 24, 1949	45.00

DERMATOLOGY AND SYPHILOLOGY

Cook County Graduate School of Medicine, Chicago	Clinical Dermatology	2 weeks, full time, every 2 weeks	75.00
New York Polyclinic Medical School and Hospital, New York	Dermatology and Syphilology	6 weeks, part time, first of any month	100.00
	Dermatology and Syphilology	3 months, part time, first of any month	200.00

DIABETES

New York Postgraduate Medical School, New York	#313—Diabetes Mellitus, Nephritis and Hypertension	Thursdays, part time, Jan. 6-Feb. 24, 1949	45.00

POSTGRADUATE COURSES

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

ELECTROCARDIOGRAPHY

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
University Extension, University of California Medical School, Los Angeles, in cooperation with University Medical Extension, University of California, San Francisco At: University Extension, Los Angeles	Electrocardiography	3 weeks, part time, Jan. 5-20, 1949	$ 25.00

ENDOCRINOLOGY

New York Postgraduate Medical School, New York	#222—Gynecological Endocrinology	24 sessions, part time, beginning Jan. 4, 1949	150.00
	#309—Diseases of the Thyroid and Other Endocrine Glands and Disorders of Nutrition	Fridays, part time, Jan. 7-Feb. 25, 1949	45.00

ENDOSCOPY

Cook County Graduate School of Medicine, Chicago	Practical Cystoscopy	10 days, part time, every two weeks	150.00
New Pork Postgraduate Medical School, New York	#211—Cystoscopy and Endoscopy	15 sessions, part time, beginning Jan. 3, 1949	75.00

GASTROENTEROLOGY

New York Polyclinic Medical School and Hospital, New York	Gastroenterology	6 weeks, part time, beginning Jan. 2, 1949	50.00
	Gastroenterology	3 months, part time, beginning Jan. 2, 1949	75.00
	Proctology, Gastroenterology, Operative Proctology (Cadaver)	6 weeks, part time, beginning Jan. 2, 1949	200.00
New York Postgraduate Medical School, New York	#311—Gastroenterology	7 weeks, part time, Jan. 5-Feb. 23, 1949	45.00

HEMATOLOGY

Graduate Division of the School of Medicine, University of Southern California, Los Angeles At: Los Angeles County Hospital	Course No. 738—Hematology	12 weeks, part time, beginning Jan. 3, 1949	50.00
New York Postgraduate Medical School, New York	#306—Clinical Hematology	Wednesdays, part time, Jan. 5-Feb. 23, 1949	30.00

INTERNAL MEDICINE

Graduate Division of the School of Medicine, University of Southern California, Los Angeles	Clinical Review of Internal Medicine (Course No. 730)	12 weeks, full time, Jan. 3, 1949	250.00
At: Los Angeles County Hospital	Internal Medicine (Course No. 731)	9 months, full time, Jan., 1949	750.00
Tufts College Medical School, Postgraduate Division, Boston At: Pratt Diagnostic Hospital	Internal Medicine (as related to Basic Sciences)	8 weeks, Jan. 3-Feb. 26, 1949	200.00 Tuition 5.00 Reg. Fee

POSTGRADUATE COURSES

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
New York Postgraduate Medical School, New York	#300—Seminar in Internal Medicine	8 weeks, full time, Jan. 3-Feb. 25, 1949	$300.00
	#307—Problems in Diagnosis (Internal Medicine)	Mondays, part time, Jan. 3-Feb. 21, 1949	45.00
	#310—Diseases of the Liver and Biliary Tract	Wednesdays, part time, Jan. 5-Feb. 23, 1949	30.00
	#315—Psychological Aspects of Internal Medicine	Fridays, part time, Jan. 7-Feb. 25, 1949	25.00
	#319—Peripheral Vascular Diseases	Tuesdays, part time, Jan. 4-Feb. 15, 1949	30.00
MEDICINE, GENERAL			
Lahey Clinic, Boston At: Clinic Auditorium	Medical Clinics	Tuesdays and Thursdays throughout the entire year	Not given
	Two-Hour Clinical Symposium on General Clinical Subjects	Wednesday nights, throughout the year, beginning Oct. 1, 1948	Not given
New York Polyclinic Medical School and Hospital, New York	Course for General Practitioners	6 weeks, full time, beginning Jan. 2, 1949	100.00
	Course for General Practitioners	3 months, full time, beginning Jan. 2, 1949	200.00
NEUROLOGY AND PSYCHIATRY			
Graduate Division of the School of Medicine, University of Southern California, Los Angeles At: Los Angeles County Hospital	Course No. 742—Psychosomatic Medicine	12 weeks, part time, beginning Jan. 5, 1949	50.00
NUTRITION			
New York Postgraduate Medical School, New York	#309—Diseases of the Thyroid and Other Endocrine Glands and Disorders of Nutrition	Fridays, part time, Jan. 7-Feb. 25, 1949	45.00
OBSTETRICS AND GYNECOLOGY			
New York Postgraduate Medical School, New York	#200—Seminar in Gynecology	2 months, full time, beginning Jan. 3, 1949	300.00
	#203—Diagnosis and Office Treatment (Gynecology)	15 sessions, part time, beginning Jan. 3, 1949	75.00
	#223—Vaginal Cytology (Specialists)	8 weeks, part time, beginning Jan. 4, 1949	100.00
University of Oklahoma School of Medicine and University Hospitals, Oklahoma City At: State Medical School of Oklahoma, Oklahoma City	Postgraduate Course in Pediatrics and Postgraduate Course in Obstetrics	5 days, quarterly	None
OPHTHALMOLOGY			
Graduate School of Medicine, University of Florida, Jacksonville At: Miami Beach, Florida	Midwinter Seminar in Otolaryngology and Ophthalmology	6 days, full time, Jan. 10-15, 1949	Not given

POSTGRADUATE COURSES

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
Harvard Medical School, Courses for Graduates, Boston	Clinical Ophthalmology	1 month, full time, Jan. 3-29, 1949	$150.00
At: Harvard Medical School and Massachusetts Eye and Ear Infirmary			75.00 Tuition
Tufts College Medical School, Postgraduate Division, Boston	Ophthalmoscopy	1 month, part time, Jan. 10-Feb. 4, 1949	5.00 Reg. Fee
At: Boston Dispensary	Clinical Otolaryngology and Ophthalmology	6 weeks, full time, Jan. 2, 1949	100.00
New York Polyclinic Medical School and Hospital, New York	Clinical Otolaryngology and Ophthalmology	3 months, full time, Jan. 2, 1949	
	Ophthalmology	6 weeks, part time, Jan. 2, 1949	150.00
	Ophthalmology	3 months, part time, Jan. 2, 1949	50.00
	Ophthalmology, Including Operative Ophthalmology (Cadaver) and Refraction	3 months, part time, Jan. 2, 1949	75.00
			275.00
	Ophthalmology and Otolaryngology, Including Cadaver Courses and Refraction	3 months, full time, Jan. 2, 1949	600.00
ORTHOPEDIC SURGERY			
New York Postgraduate Medical School, New York	#500—Seminar in Orthopedic Surgery (Specialists)	10 days, full time, Jan. 3-14, 1949	125.00
OTOLARYNGOLOGY			
Graduate School of Medicine, University of Florida, Jacksonville At: Miami Beach, Florida	Midwinter Seminar in Otolaryngology and Ophthalmology	6 days, full time, Jan. 10-15, 1949	Not given
New York Polyclinic Medical School and Hospital, New York	Clinical Otolaryngology	6 weeks, part time, Jan. 2, 1949	75.00
	Clinical Otolaryngology	3 months, part time, Jan. 2, 1949	100.00
	Clinical Otolaryngology and Ophthalmology	6 weeks, full time, Jan. 2, 1949	100.00
	Clinical Otolaryngology and Ophthalmology	3 months, full time, Jan. 2, 1949	150.00
	Ophthalmology and Otolaryngology Including Cadaver Courses and Refraction	3 months, full time, Jan. 2, 1949	600.00
PATHOLOGY			
Cook County Graduate School of Medicine, Chicago	Gross Pathology	1 month, full time, every month	150.00
	One Month Course—Surgical Pathology	4 weeks, full time, every month	250.00
	Two Weeks Course in Surgical Pathology	2 weeks, full time, every two weeks	150.00

POSTGRADUATE COURSES

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

PEDIATRICS

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
Harvard Medical School, Courses for Graduates, Boston At: Children's Hospital	Pediatrics	4 months, full time, Jan. 3-Apr. 28, 1949	$500.00
New York Medical College, Flower and Fifth Avenue Hospitals, New York	Clinical Pediatrics Clinical Pediatrics	1 month, all year, full time 3 months, all year, full time	150.00 250.00
New York Postgraduate Medical School, New York	#410—Clinical Pediatrics	4 weeks, full time, Jan. 3-29, 1949	150.00

POLIOMYELITIS

Georgia Warm Springs Foundation, Warm Springs	Care of Acute and Convalescent Poliomyelitis	1 week, Jan. 3, 1949	None

PROCTOLOGY

Tufts College Medical School, Postgraduate Division, Boston At: Boston Dispensary and Associated Clinics	Proctology III (Proctology I is prerequisite to Proctology III)	1 month, part time, Jan. 3-29, 1949. (Course may be taken 6 days a week for one month or 3 days a week for 2 months)	100.00 Tuition 5.00 Reg. Fee
New York Polyclinic Medical School and Hospital, New York	Proctology	6 weeks, part time, Jan. 2, 1949	75.00
	Proctology, Gastroenterology, Operative Proctology (Cadaver)	6 weeks, part time, Jan. 2, 1949	200.00

PUBLIC HEALTH

Ohio State Department of Health, Columbus At: Oberlin, Ohio	Field Training, Orientation and Public Health Administration	1 month, special arrangement	None

RADIOLOGY

Cook County Graduate School of Medicine, Chicago	Clinical Roentgenology	2 weeks, full time, 3rd Monday of each month	70.00
	#1—Diagnostic Roentgenology	2 weeks, full time, 1st Monday of each month	125.00
	One Month Course—Clinical Roentgenology	4 weeks, full time, 3rd Monday of each month	125.00
	Radiation Therapy	2 weeks, full time, every two weeks	110.00
	Radiation Therapy	4 weeks, full time, every month	200.00
	Radiation Therapy	2 months, full time, every month	300.00
Harvard Medical School, Courses for Graduates, Boston At: Children's Hospital	Pediatric Radiology (limited to physicians who have a working knowledge of Radiology)	1 month, full time, monthly	150.00
At: Massachusetts General Hospital	General Radiology (limited to physicians who have a working knowledge of Radiology)	1 month, full time, monthly	150.00

POSTGRADUATE COURSES

Selected Postgraduate Continuation Courses for Practicing Physicians—Beginning January 1, 1949.

Institution	*Title of Course*	*Schedule of Course*	*Registration Fee and/or Tuition*
At: Peter Bent Brigham Hospital	General Radiology (limited to physicians who have a working knowledge of Radiology)	1 month, full time, monthly	$150.00
New York Medical College, Flower and Fifth Avenue Hospitals, New York	Diagnostic Roentgenology	6 weeks, half time, every 6 weeks except during July and Aug.	150.00
New York Polyclinic Medical School and Hospital, New York	Radiology	3 months, full time, Jan. 2, 1949	300.00
	SURGERY		
New York Polyclinic Medical School and Hospital, New York	Clinical Surgery	6 weeks, full time, Jan. 2, 1949	100.00
	Combined Surgical Course Including Cadaver Surgery and Gynecology	3 months, full time, Jan. 2, 1949	350.00
	THERAPY		
Cook County Graduate School of Medicine, Chicago	Radiation Therapy	2 weeks, full time, every two weeks	110.00
	Radiation Therapy	4 weeks, full time, every month	200.00
	Radiation Therapy	2 months, full time, every month	300.00
	UROLOGY		
New York Hospital (James Buchanan Brady Foundation), New York	Postgraduate Course in Urology	31 weeks, full time, 6 months or longer, Jan. 1, 1949 (31 weeks, full time, for veterans; 6 months or longer for others)	500.00
New York Postgraduate Medical School, New York	#1010—Advanced Course in Urology for Specialists	2 months, full time, Jan. 3-Feb. 26, 1949	300.00

ADDRESSES OF SCHOOLS LISTED

CALIFORNIA

Graduate Division of the School of Medicine, University of Southern California, Los Angeles 7, California.
University Extension, University of California Medical School, Los Angeles 24, California.
University Medical Extension, University of California, San Francisco 22, California.

FLORIDA

University of Florida Graduate School of Medicine, 2033 Riverside Avenue, Jacksonville, Florida.

GEORGIA

Georgia Warm Springs Foundation, Warm Springs, Georgia.

ILLINOIS

Cook County Graduate School of Medicine, 427 South Honore Street, Chicago 12, Illinois.

MASSACHUSETTS

Harvard Medical School, Courses for Graduates, 25 Shattuck Street, Boston 15, Massachusetts.
Lahey Clinic, 605 Commonwealth Avenue, Boston 15, Massachusetts.
Tufts College Medical School, Postgraduate Division, 30 Bennet Street, Boston 11, Massachusetts.

NEW YORK

New York Hospital (James Buchanan Brady Foundation), 525 East 68th Street, New York 21, New York.
New York Medical College, Flower and Fifth Avenue Hospitals, 20 East 106th Street, New York 29, New York.
New York Polyclinic Medical School and Hospital, 345 West 50th Street, New York 19, New York.
New York Postgraduate Medical School, 303 East 20th Street, New York 3, New York.

OKLAHOMA

University of Oklahoma School of Medicine and University Hospitals, 800 Northeast 13th Street, Oklahoma City 4, Oklahoma.

OHIO

Ohio State Department of Health, 306 Ohio Depts. Building, Columbus 15, Ohio.

PENNSYLVANIA

University of Pennsylvania Graduate School of Medicine, 237 Medical Laboratories, Philadelphia 4, Pennsylvania.

Leaves from a Doctor's Diary

By Maurice Chideckel

November 1 . . . Man's pretense to be a reasoning animal is a misrepresentation at times, a forgery, a lie, if you please. Apropos of the case of intestinal amebiasis, treated with emetine by capable Dr. Gibbons. The patient died of a severe myocarditis. I don't know whether liver involvement is or is not present in such cases. Nor do I know whether it was the toxicity of the emetine that killed the patient. Perhaps it were better to have used a nontoxic drug, say, chloroquine, instead of emetine.

But was it sensible, was it ethical on the part of Dr. John O'Higgins to tell a neighbor of the dead man that it was bad treatment that killed the patient? The two bully brothers of the deceased threatened to make mincemeat out of Dr. Gibbons. And Dr. O'Higgins is a dignified, sensible looking gentleman.

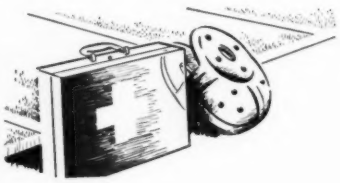
* * *

November 2 . . . Boldly planned and triumphantly achieved by refugee Dr. Kaltenheimer, Mrs. Snellenburger, also a refugee, simply had to have a baby, or her Americanized husband threatened to leave her in the cold. The husband's sperm, to use the expression of Dr. Kaltenheimer, "is very dead." Sly Dr. Colton suggested artificial insemination. "Goot, so goot," joyously exclaimed the imported medico. It was done unbeknown to hubby. And the lady conceived, and she was with child and gave birth to it—a boy.

How adoringly "papa" gazed at his "offspring." And he spoke. "Did you ever, ever, see such a striking resemblance of father to son?—Just me in miniature." Dr. Colton pressed the poor refugee physician for details. Where did he get the donor? How much was he paid? Kaltenheimer shrugged his shoulders. This, and nothing else. Then Dr. Colton passed judgment: "It was not artificial insemination." Oh, well, could be.

* * *

November 6 . . . Colleague Slade and I walked around the clinic and discussed the cases of two young men, two tragedies. One underwent a thorough physical in the morning for a football team, and was declared a fine specimen. After half an hour of strenuous exercise he dropped dead. Death for this boy of 24 proved to be due to atherosclerosis of both coronaries.



The other case was that of a young man who underwent a premarital examination and was given a clean bill of health. He went on his honeymoon and after coitus with the bride his headache became so violent that he was brought home where he died. Death was due to intracranial hemorrhage, caused by a tiny aneurysm in the circle of Willis. "There is your value of our physical," gloomily spoke my companion.

A soft hand was laid on his shoulder, the one belonging to Mrs. Paperman. "Just coming from your office. You must tell me what to do. My husband keeps on talking in his sleep a whole night, and I can't close my eyes. What am I to do?" Angrily Dr. Slade answered: "Give him a chance to say a few words during the day and he'll keep quiet during the night." Said she: "Will that really help? Oh, thank you."

November 10 . . . Says Dr. Random to me: "Have you seen that rare case of complete atresia of the esophagus with the lower end communicating with the trachea near the bifurcation? Did you notice the upper segment ending in a blind pouch? Well, I am to ligate the fistula. I'll try to collect the fee in advance. Why? Well, he may not survive the ligation. He. . . ." His wife approached him. "Nice girl, if she would not be so talkative. Talks and talks and says absolutely nothing." "Oh, darling," she began, "I was over at the fashion show. It's sublime, wonderful. It absolutely made me speechless." From him: "Well, if it made you speechless, I'll buy the whole outfit, regardless of price. Wonderful investment." Her eyes spat fire. "Brute," she screamed, "I never want to see you again," and away she rushed. He smiled. "She'll come back. Anything to make her speechless, if only for a week, is worth the price."

* * *

November 12 . . . Told John Fairchild, 88, that he was suffering from pulmonary tuberculosis in advanced stage. One more proof of the laity's misconception that it never occurs in the aged. The old gentleman fell to thinking. "So I can't get married. Or can I? Bad. She'll be disappointed. Planned a honeymoon. Sorry I came here."

Not Mrs. Pratt, whose husband, 79, suffers from thromboembolism. I warned her that the elderly man must stay out of bed in order to avoid a lethal catastrophe. I explained to her the danger of the absence of leg movement and the obstruction to the blood flow by flexion of the thighs. "Gosh," she addressed herself to her woman companion in the hall, "I am glad I came here. I thought he must stay in bed, so I made him walk. Now, to bed he goes. What do you think of that dope, the doctor? Does he think I married that antique for love?" Said the companion: "Dope is right."

November 15 . . . Dr. Lewis Elmer's life is devoted to the exploration of the abnormal mind. He can interpret the fears, the resolves, the deprevations, the laughter, the fury of the insane. The vague, the furtive bits of knowledge possessed by the mentally unbalanced, he can translate and form into impressions. As an experiment, he tried to persecute one inmate of a mental hospital. That inmate loved and adored Dr. Elmer. This, he explained to me, is the affinity between the hunter and the hunted.

The doctor began to devote a great deal of his time to that inmate of 21, who was illegitimate and the son of a prostitute. He came to the conclusion that the boy was never insane, only a lonely, misguided child who had fallen in bad company. Himself childless, the doctor adopted him, took him to his house, educated him. It was a happy family circle, for the former inmate was very ingratiating and one can say charming. And suddenly, because of an imaginary affront, he turned savagely on his benefactor. Dr. Elmer is in the hospital with four broken ribs and a fractured lower maxilla. Back in the asylum the inmate has only one wish: Finish his job. "Who did he think I was? A lunatic?" Unable to speak, Dr. Elmer wrote on a paper: "You cannot universalize the abnormal."

The normal, as said Jacobi long ago, is a myth. Patient Ludwig demanded that I give him back the medical fee he paid me after an examination, because he decided not to take the prescription. Is he normal? And when I granted his request, am I normal?

* * *

November 18 . . . There he stood, Claude Whitaker, in a dress shop. It was a stormy discussion, because of his case. Rhetorical Lucille, our feminine cardiologist wrote these words in her report: "He attracted the first bolt of lightning from the storm clouds of progressive medicine."

Claude's was an everyday case of coronary thrombosis. As his prothrombin time was found normal, he was to be given dicumarol and heparin. Dr. Robert forcibly took away the medication from the hands of the interne. He proclaimed what we already heard and knew, that in the episodes of coronary occlusion, there is rather a tendency to bleeding into the wall of the artery, which causes the thrombosis. Hence instead of an anticoagulant, we must administer a drug that will promote coagulation. Claude Whitaker was given dicumarol and there he was. Orthopedic surgeon G. and I wanted to go in and talk about his condition. But he had a customer, one of those females that look blissfully ignorant and incurably vain. We saw him take out one dress after another, and we decided to stop in on our way back.

More than an hour later, the same customer was still there. The store looked like the aftermath of a Russian pogrom and Claude was on the point of collapse. The counters were piled up with dresses, all boxes empty, with the exception of one on the shelf. "Well," she finally said, "I really didn't come to buy anything. I had a couple of hours to spend, so I thought I'd look at some dresses. You don't mind, do you?" Dr. G. suggested that I break her nose and then take her to his office and he'd set it. Claude was still smiling. "In this case, lady, will you kindly permit me to show you the last dress we have here. Only one left that you haven't seen." She benevolently condescended. Believe it or not, that dress she bought, actually paid for, and left with it.

November 22 . . . Some insignificants.

Two middle-aged women left the clinic angrily, when the examiner insisted that they state their correct age. So said Anatole France once, "I'd rather ask an old man when he expected to die than an old woman when she was born."

An old man laughed hilariously when Dr. Gallagher told him to stop drinking twelve cups of coffee a day, because coffee is a slow poison. "Very, very slow," he spoke aloud. "Been drinking it for sixty-eight years."

Because of the tannin in tea which may be a cause of constipation, Russian born Fyodor Malinov was asked how much tea he drank in a day. Answer: Twenty-two saucers.

Samuel Gellerman is in a hospital with a burned face, because of lye thrown at him by his sweetheart, when she learned of a date he had with another young woman. So said William Congreve:

Heaven has no rage like love to hatred turned,
Nor Hell a fury like a woman scorned.

Pious Sol Roberts with youth far behind him, met beautiful Mathilde in my waiting room. He offered his life for one kiss from her and she refused to sell her kisses so cheap.

And from all the men and women I meet and treat, I have learned that man's mind is the most unexplored region, and will remain obscure until the end of time, for there is no such thing as the embodiment of intellect. The man, even the learned man, who believes in the self sufficiency of his own thought is deceiving himself. The man with a complete mind is as yet unborn, and never will be.

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*Beame, A. J., and Endicott, E. T., Histologic changes in the livers of patients with cirrhosis treated with methionine, Gastroenterology 9:718-735 (Dec.) 1947.

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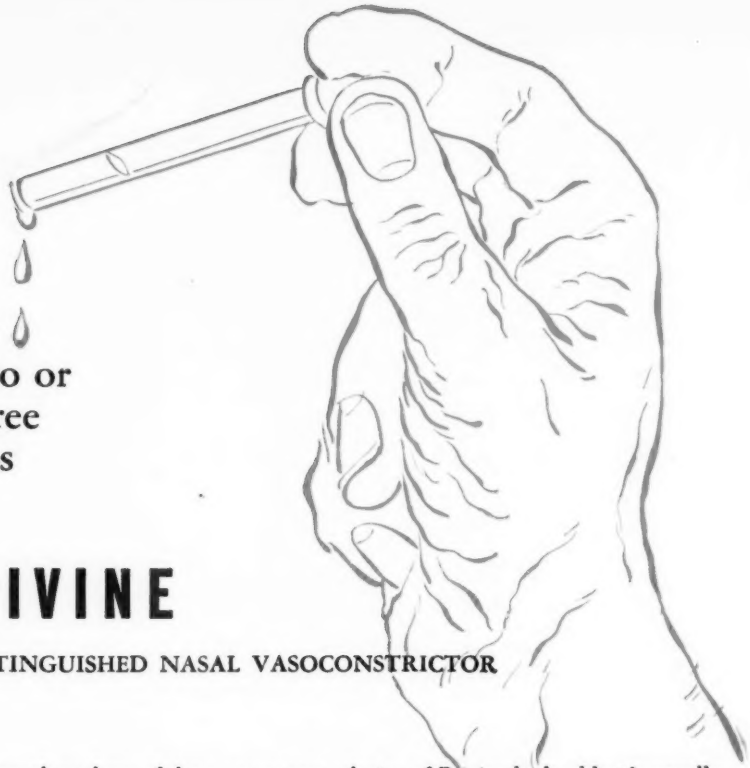

only two or three drops

PRIVINE

A DISTINGUISHED NASAL VASOCONSTRICTOR

high potency Only two or three drops of the 0.05 per cent solution of Privine hydrochloride usually give prompt and complete relief of nasal congestion and hypersecretion.

prolonged action The effect of each application of Privine provides two to six hours of nasal comfort, thus avoiding the inconvenience of frequent re-application.

bland and non-irritating Privine is prepared in an isotonic aqueous solution buffered to a pH of 6.2 to 6.3. Artificial differences in osmotic pressure between solution and epithelium are avoided; stinging and burning are usually absent.

relatively free from systemic effects Although a sedative effect is occasionally noted in infants and young children – usually after gross overdosage – Privine is generally free of systemic effect. The absence of central nervous stimulation permits the use of Privine before retiring without interfering with restful sleep.

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Privine 0.05 per cent for all prescription purposes; 0.1 per cent strength reserved for office procedures.

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